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**IMPACT OF STATIC FLEXION DURATION ON THE DEVELOPMENT OF A
NEUROMUSCULAR DISORDER OF THE LUMBAR SPINE**

A Thesis

Submitted to the Graduate Faculty of the
Louisiana State University and
Agricultural and Mechanical College
in partial fulfillment of the
requirements for the degree of
Master of Science in Biological and
Agricultural Engineering

in

The Department of Biological and Agricultural Engineering

By

Rebecca Victoria LaBry
B.S., Louisiana State University, 2003
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ABSTRACT

Many occupational groups work under adverse conditions in which their spinal columns are fully flexed under load for substantial periods of time. This study was designed to determine the response of the lumbar spine to a static load of a substantial duration under a range of load magnitudes. The impact of static loads of 20, 40 and 60N were applied over 30 min flexion, 10 min rest, and 30 min flexion (for a total of one hour exposure) followed by a 7 h rest. Lumbar viscoelastic creep (laxity) and reflex electromyographic (EMG) activity were monitored over both flexion periods and the 7 h rest period. It was found that 10 min of rest was not sufficient for complete recovery of the creep developed in the first 30 min of flexion resulting in a large cumulative creep at the end of the work-rest session. Muscle activity indicated spasms during the static flexion periods and demonstrated the development of initial and delayed hyperexcitability in each of the 3 loads. Larger magnitudes of initial and delayed hyperexcitability were observed for larger loads although the differences were not statistically significant. Thus, intense periods of static flexion will result in neuromuscular disorders regardless of load magnitude. The results of the 3:1 work-rest ratio were compared with previously obtained data of a series of short static flexion periods (1:1 ratio) of the same cumulative time to determine which can best expedite the recovery of creep. Although the valuable effects of a 1:1 work-rest ratio have been documented (Sbriccoli, 2004), a 3:1 work-to-rest duration ratio was not sufficient to attenuate or prevent the development of any of the components of a neuromuscular disorder and the associated microdamage and inflammation. In conclusion, a cumulative low back disorder was elicited from exposure to two 30 min static loads spaced by a 10 min interval.

CHAPTER 1-INTRODUCTION

A wide range of afflictions affecting the tendons, nerves, muscles, and supporting structures (i.e., intervertebral discs) that are caused or made worse by a work environment are defined as work-related musculoskeletal disorders (WMSDs) by the National Institute of Occupational Safety and Health (NIOSH). Work-related musculoskeletal disorders arise from a complex of interaction of events that accumulate over time and the widespread presence and negative consequences of WMSDs are universally recognized. Musculoskeletal disorders result in decreased worker productivity, lost time from work, temporary or permanent disability, inability to perform job tasks and an increase in worker compensation costs. This is all due to the severe and disabling symptoms, such as pain and numbness, which accompany musculoskeletal disorders (NIOSH, 1997). Aside from the adverse health effects, musculoskeletal disorders have also yielded staggering economic consequences costing an estimated \$50 billion per year in direct costs with the estimate rising to over \$1 trillion annually with the inclusion of indirect costs (National Academy of Sciences, 1999).

Musculoskeletal disorders include injuries and disorders of the back, trunk, upper, and lower extremities such as carpal tunnel syndrome, tenosynovitis, and low back pain. The following injury statistics illustrate the massive magnitude, cost, and burden of WMSDs on employers and employees and testifies to the need for intervention. The Bureau of Labor Statistics reported in 2001 that sprains and strains, most often involving the back, encompassed more than 4 out of ten injuries and illnesses that resulted in days away from work, following the trend of the previous ten years (BLS, 2003). Approximately 70 million Americans sought medical treatment for musculoskeletal disorders in 1999 with over 1 million workers taking time off from work because of work-related disorders of the lower back and upper extremities

(National Research Council, 2001). More than 30 million people have low back pain at any given time in the United States, with ten million of these experiencing chronic symptoms (Panjabi, 1996). Musculoskeletal disorders are expected to increase in the future because of the changing nature of work, the aging of the workforce, and rising numbers of women entering material handling and computer jobs (National Academy of Sciences, 1999).

Thirty percent of American workers are employed in jobs that routinely require them to perform high-risk activities associated with developing low back disorders, including heavy physical work, lifting and forceful movements, bending and twisting, vibrations and static work postures (Damkot et al., 1984; Garg and Moore, 1992; Hoogendoorn et al., 2000; Kerr et al., 2001; Kumar, 2001b; National Occupational Research Agenda, 1997; Thorbjornsson et al., 2000). Workers exposed daily to static, cyclic, and vibratory occupational activities over months and years are at risk to develop cumulative trauma disorders (CTDs) (Hoogendoorn et al, 2000; Punnett et al, 1991; Silverstein et al, 1986). A multitude of factors contribute to the development of cumulative trauma disorders including the magnitude of the load sustained, the duration of this exposure, and the amount of repetitions of the exposure (Burdorf et al., 1991; Elders and Burdorf, 2001; Guo, 2002; Hoogendoorn et al, 2000; Norman et al, 1998; Punnett et al, 1991).

Private industries along with the US government implement programs and regulations to protect and improve the health and safety of workers, some with specific control measures to facilitate prevention efforts to reduce the prevalence and costs of these types of injuries. Scheduling more frequent or longer rest breaks and rotating workers between less and more ergonomically stressful jobs are examples of administrative controls often used by industries to control worker exposure to duties that are associated with high injury rates. The multifactorial etiology of musculoskeletal disorders makes these controls difficult to implement, partly because

the individual characteristics of workers such as sex, age, anthropometry, muscle strength, muscle endurance, lifestyle, and psychological factors including motivation play a role in the development of these disorders (Radwin et al., 2002). The multitude of factors that contribute to MSDs can interact in various ways so that one factor causes one type of disorder for a particular person while it causes another in a different person (Sommerich et al., 1993). A duration formula has been developed that may predict the relationships between CTD and the magnitude of loads developed within the joint tissues, the duration over which the loads were applied, and the number of repetitions that such load over time was executed (Frazer et al., In Press). Additionally, a rest period between sequential periods of static flexion sustained over time may also play a prominent role in the development or prevention of CTD. Similarly, the overall period, in months or years, that a worker was exposed to such activity may also be incorporated as a major component of any dose-duration formula (National Research Council, 2001; Occupational Safety and Health Administration, 2000). A better understanding of the maximum human capabilities in static work postures is needed to reduce the number of low back injuries by keeping the job demands below the worker's capacity. Practical data about human capabilities in specific work situations can be used to evaluate current work environments and to develop more complete dose-duration formulas.

Biomechanics is the study of mechanics of a living body and the effects of forces on the body's tissues, fluids, or materials (Radwin et al., 2002). A conceptual model of factors that may play a role in the development of WMSDs is shown in Figure 1. The biomechanical aspects of the neuromuscular system are integral in determining function and stability of the lumbar spinal muscles and ultimately the lumbar spine. This research studies the relationship between work-

rest periods and its effect on the development of spinal instability via creep, which has been shown to cause cumulative trauma disorder of the lumbar spine.

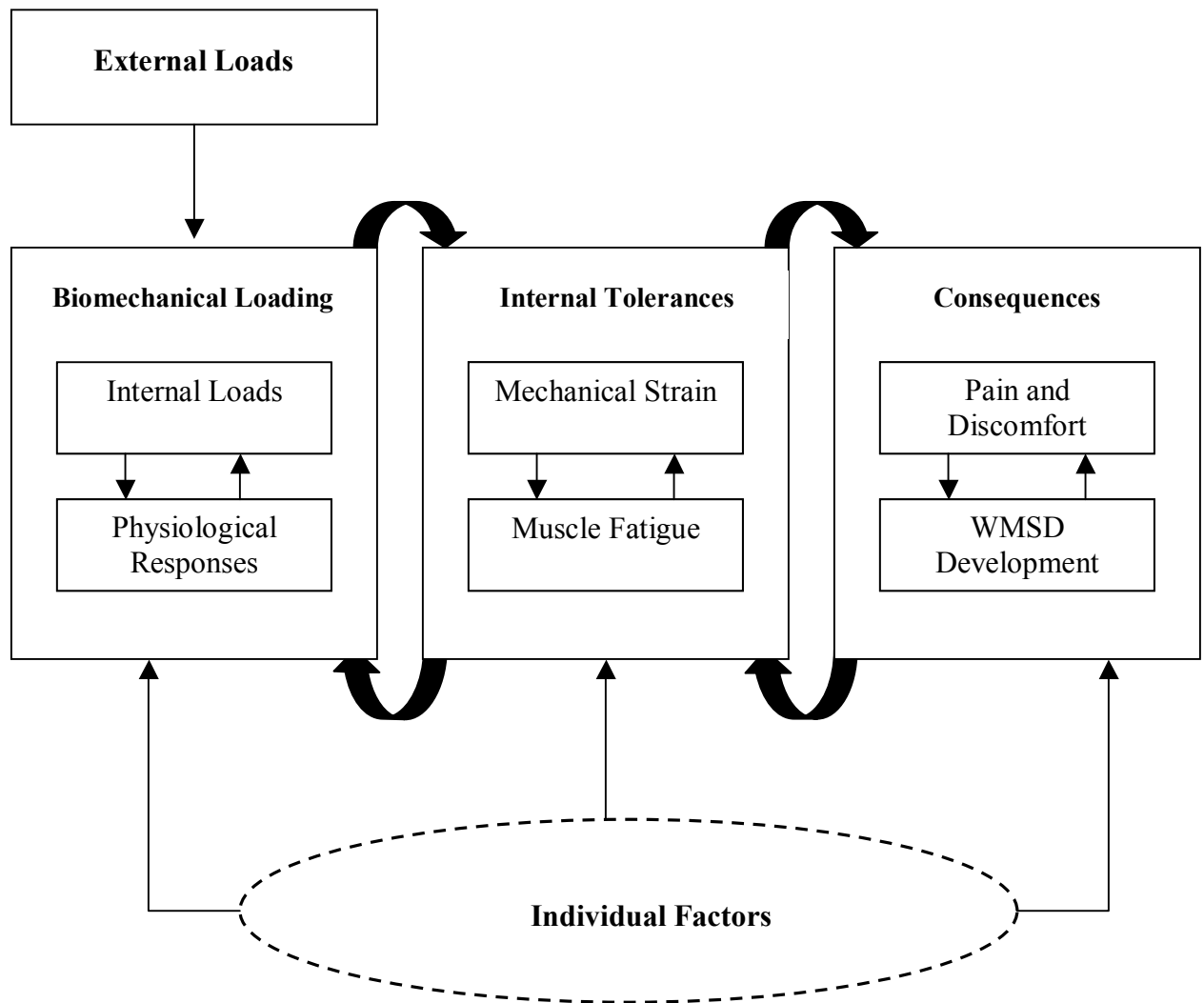


Figure 1. Conceptual model of factors that play a role in the development of work-related musculoskeletal disorders. (Modified from Radwin et al., 2002)

CHAPTER 2- BACKGROUND AND LITERATURE REVIEW

Many occupational groups such as stonemasons, bricklayers, and mechanics often expose their spine to prolonged flexion loading. There is seldom movement away from the fully flexed position and a minimal opportunity for recovery exists between episodes of work in this position (Towmey et al, 1988). Exposure to static lumbar flexion over extended periods of time may cause a cumulative trauma disorder (CTD) in workers that are continuously exposed to these conditions. The spine becomes predisposed to pain and/or injury with cumulative load exposure (Kumar, 2000a).

2.1 Anatomy and Physiology of the Lower Back

An understanding of the basic anatomical and physiological properties of the spine's structure and its constituents is essential to analyze the effects of static load on viscoelastic tissue behavior and muscle activity.

2.1.1 Vertebral Column

The vertebral column serves as the central skeletal axis of the body. It is a segmented, jointed, flexible rod-like structure which protects the spinal cord and spinal nerves, supports the weight of the body, plays an important role in posture and locomotion, and provides axial support for the limbs. The vertebral column consists of a series of 33 vertebrae arranged in 5 regions: 7 cervical, 12 thoracic, 5 lumbar, 5 sacral, and 4 coccygeal. The cervical vertebrae are located at the top of the spinal column and form a flexible framework for the neck and support the head. The next twelve vertebrae form the thoracic region of the column and serve as a rear anchor of the rib cage. The five lumbar vertebrae are the largest vertebrae in the spinal column. These vertebrae support most of the body's weight and serve as an attachment point for many of the back muscles. The sacrum is a triangular bone located just below the lumbar vertebrae and

consists of 5 sacral vertebrae fused together. The coccyx, consisting of 4 fused coccygeal bones, is the bottom of the spinal column.

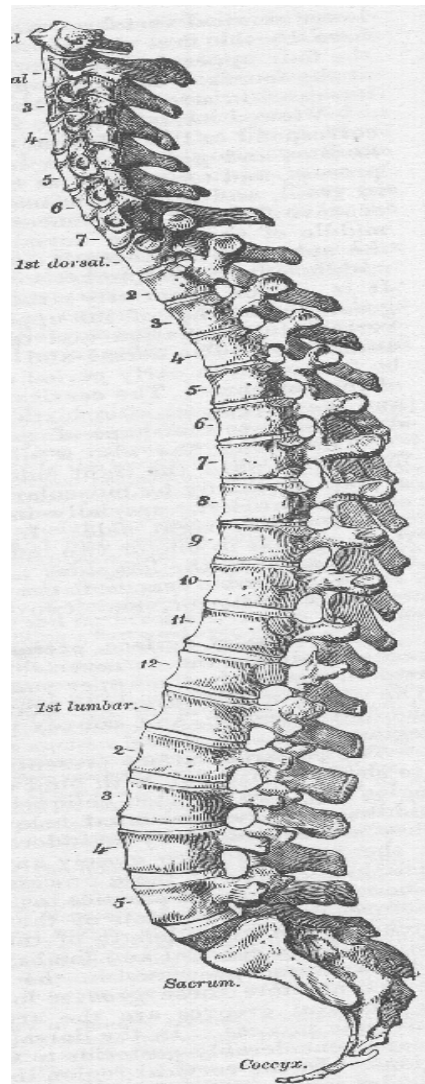


Figure 2. Human Spinal Column (Gray, 1974)

2.1.2 Intervertebral Discs

The intervertebral discs provide strong attachments between the vertebral bodies, act as shock absorbers, and provide the main strength and stiffness of the motion segment of the spine. Each intervertebral disc is made up of the nucleus pulposus and an annulus fibrosis. The nucleus pulposus forms the central core of an intervertebral disc and contains a gelatinous substance.

Tough fibrocartilagenous fibers known as the annulus fibrosis surround the nucleus pulposus and form the circumference of the intervertebral disc.

2.1.3 Ligaments and Tendons

Tendons and ligaments are fibrous connective tissues. Ligaments join bones and provide stability to the joints. Tendons help execute joint motion by transmitting mechanical forces from muscle to bone whereas the mechanical role of ligaments is to transmit forces from one bone to another. However, as passive tissues they cannot actively contract to generate forces.

2.1.4 Supraspinous Ligament

The supraspinous ligament is a single, long vertical fibrous band passing over and attached to the tips of the spinous processes thereby connecting vertebrae from the seventh cervical to the sacrum. It is thickest and broadest in the lumbar region. The most superficial fibers of this ligament connect three or four vertebrae, the deeper fibers pass between two or three vertebrae, and the deepest connect adjacent extremities of neighboring vertebrae (Gray, 1974). The supraspinous-interspinous ligament segments are the first ligamentous tissues to become stressed with forward bending of the lumbar spine. (Adams et al, 1980).

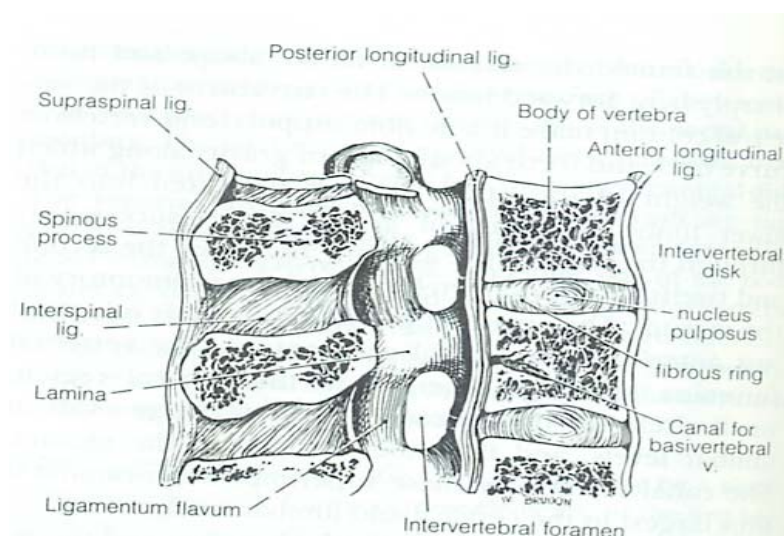


Figure 3. Ligaments of the Spine (Woodburne and Burkel, 1988)

2.1.5 Fascia

This term refers to flat layers of fibrous connective tissues usually in the form of a membranous sheet. Its function is to separate different layers of tissue as well as enveloping the muscles, bones and joints. The thoracolumbar fascia consists of three layers of fascia that envelop the muscles of the lumbar spine, effectively separating them into 3 compartments.

2.1.6 Multifidus Muscle

The multifidus is the largest and most medial of the lumbar paraspinal muscles. It is found on both sides of the spinous processes from the sacrum to the axis and exhibits a constant pattern of attachments caudally. Each bundle of the multifidus originating from one vertebra has termination on the second to fourth vertebrae below. It functions as a vital segmental stabilizer as well as allowing for lateral flexion, rotation, and extension.

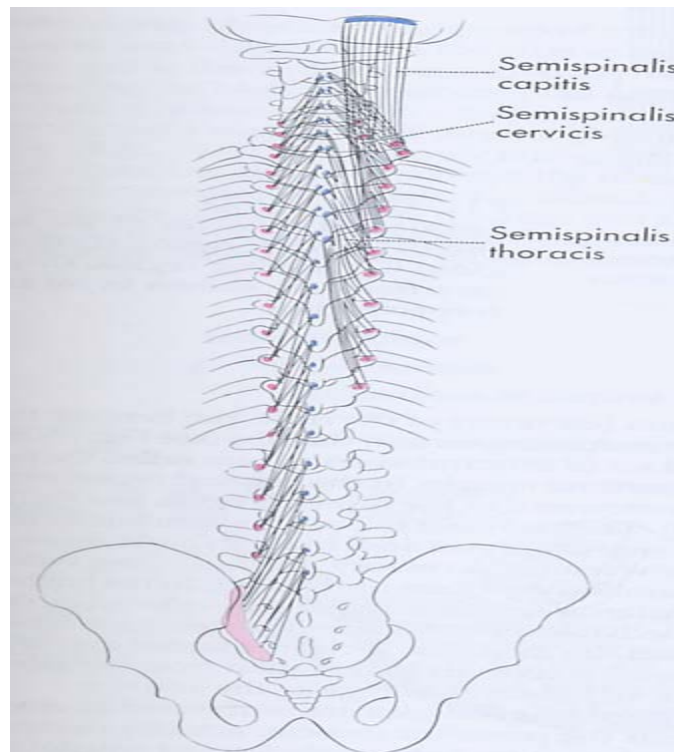


Figure 4. Multifidus muscle attached to vertebrae are shown on the left (Rosse and Gaddum-Rosse, 1997)

2.1.7 Mechanoreceptors

Mechanoreceptors are cells specialized to transduce mechanical stimuli such as pressure or stretching and relay the acquired information to the nervous system (Dorland, 1994). They give us the sense of proprioception, or the ability to unconsciously monitor the position of our muscles, bones, and joints.

Only type II and type III mechanoreceptors are known to be present in the supraspinous ligament (Hirsch et al., 1963; Rhalmi et al., 1993; Yahia et al., 1988; Yahia and Newman, 1991). Type II mechanoreceptors are low threshold and rapid adaptors. They signal the initiation and termination of a stimulus to the central nervous system. The sensitivity of type II mechanoreceptors to minor changes in tension allows them to provide continuous signal in ligament strain. Type III receptors are slow adaptors with high thresholds that only provide signals when the ligament is experiencing extreme amounts of strain. The supraspinous ligament is also embedded with free nerve endings that provide long-lasting information on the deformation of the tissues and pain sensation (Rhalmi et al, 1993; Yahia and Newman, 1993). The timing and intensity of tissue deformation is monitored by all of the receptors collectively (Petrie et al., 1998).

2.2 Electromyography

Electromyography (EMG) is the study of muscle function through analysis of the electrical signals emanated during muscular contractions. In order to understand how this is accomplished, an understanding of how a muscle functions and the components of an EMG system are necessary.

2.2.1 Basic Muscle Functions

A motor unit is the single smallest controllable muscular unit and functional unit of striated or skeletal muscle. The nerve cell body, its axon, terminal branches, and all muscle fibers supplied by these branches form a motor unit. One motor unit may have from 3 to 2000 fibers. Motor units controlling fine movements and adjustments typically have less than 10 muscle fibers per unit, whereas motor units controlling coarse actions have hundreds to thousands of muscle fibers per unit. (Ganong, 1981) Under normal circumstances, an action potential descends the nerve axon and activates all the muscle fibers of the motor unit. (Paton and Wand, 1967) A single muscle fiber will never contract individually in accordance with the all or none rule, but only together with the rest of the fibers innervated by the same cell.

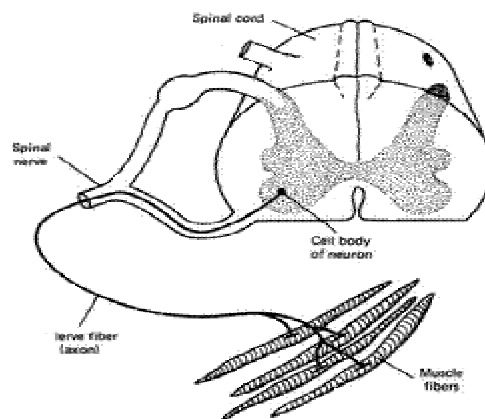


Figure 5. Components of the motor unit (Basmajian and Deluca, 1986)

The signal propagates along the muscle fiber, which generates ion movement across the muscle cell membrane. An electric field is produced and can be detected by electrodes near the activated muscle fibers. The motor unit action potential (MUAP) is the summation of the individual muscle fiber action potentials. Located within a specified recording area, there may be muscles

fibers from many motor units. The algebraic summation of all the detected signals forms the EMG signal.

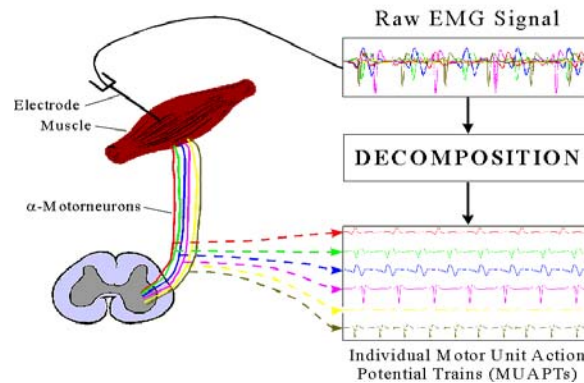


Figure 6. Collection of Motor Unit Action Potential (MUAP) and resulting EMG signal (Neuromuscular Research Center, 2002)

2.2.2 Recording Techniques

An EMG collection system must consist of electrodes, amplifiers, filters and an acquisition device. Electrodes serve as the site of connection between the body and the collection system. Electrodes are available in both the invasive (i.e., wire and needle) and non-invasive (i.e., surface) variety; however, wire electrodes are the electrodes of choice for this study because they allow for the examination of deep muscles. Wire electrodes are typically small-diameter, flexible, non-oxidizing wires, which are inserted into the muscle and insulated except for 1-2 mm at the tip of the wire. The un-insulated portion of the wire serves as the detection area. The amplifier performs several important functions including isolation between the signal source and the recording instrumentation, current to voltage conversion and voltage gain and noise reduction. Filters serve to eliminate any noise, or unwanted signal detected alongside the wanted signal. The acquisition device may be an oscilloscope or computer, which allows for the observation and recording of data, respectively. Each of these components is designed to

maximize the amount of information obtained from the EMG signal and to minimize the amount of contamination from electrical noise (Acierno et al., 1995).

2.3 Cumulative trauma load-tolerance model

Load is used to describe kinetic physical stresses (force) acting on anatomical structures within the body. Loads may originate from the external environment or result from voluntary or involuntary actions of the individual. External loads are transmitted through the limbs and body structures to create internal loads on tissues and anatomical structures. The term tolerance describes the physical ability of structures within the body to withstand loading. Cumulative trauma is the result of accumulated effects of brief external loads which acting alone are insufficient to exceed tissue tolerances as opposed to acute trauma injuries which arise from a single identifiable event. The internal tolerances of the tissues will be exceeded over time as the loading accumulates through repeated exposures or if the exposure is maintained for a significantly long duration (Radwin et al, 2002). Internal tissue tolerances may become lowered through repetitive or sustained loading.

The development of cumulative trauma disorders is influenced by magnitude, frequency and duration (Hoogendoorn et al, 2000; Norman et al, 1998). Magnitude quantifies the amplitude of the force applied. Repetition corresponds to the frequency or rate at which a physical stress factor repeats. Duration is the time of exposure to a physical stress factor. Duration may indicate the length of a single exposure or the cumulative exposure over a day or years. Similarly, rest may refer to the time between single exposures or between days of work.

the feline could be extrapolated to humans with an appropriate scaling for size and compensation for intrinsic differences.

2.5 Literature Review

A predominant cause of low back pain is spinal instability. Panjabi describes spinal stability as centering on the interrelationships, disposition, and alignment of the vertebrae in both static and dynamic phases (Panjabi, 1993). A 3-component system made up of a passive subsystem (i.e., ligaments, discs), an active subsystem (i.e., muscles), and the neural-feedback subsystem forms the stabilizing system of the spine (Panjabi, 1992). The primary structure responsible for the stability of the lumbar spine is the musculature associated with it (Granata and Marras, 1995; Kaigle et al., 1995; McGill and Norman, 1986; Panjabi, 1992; Pope et al., 1986; White and Panjabi, 1978), whereas the passive viscoelastic structures (ligaments, discs, and capsules) function as secondary stabilizers (Crisco et al., 1992; McGill and Norman, 1986; Posner et al., 1982; Teo and Ng, 2001; White and Panjabi, 1978). An intact spinal column is capable of carrying only a small load, approximately 90N or 20 pounds, without buckling or mechanical instability, whereas with the addition of the spinal muscles for stabilization, a healthy person is able to carry substantially higher loads (Cholewicki and McGill, 1991). Although the role of muscles in spine stabilization is fundamental, the neuromuscular control system is the most important component of the stabilizing system. The passive system establishes the stability requirements and the neural-feedback system recruits the active subsystem to meet the demand through activation of the spinal muscles.

The knee, shoulder, elbow, ankle joints, and spine of humans and animals are stabilized by a ligamento-muscular reflex arc. Spinal musculature and viscoelastic tissues work synergistically. Sensory receptors in the viscoelastic tissues trigger a reflex contraction of the

appropriate muscles in the spine (Guanche et al., 1995; Hirokawa et al., 1991; Knatt et al., 1995; Lewis et al., 1996; Phillips et al., 1997; Solomonow et al., 1998; Solomonow et al., 1996; Solomonow et al., 1987; Stubbs et al., 1998; Williams et al., 2000).

Biological tissues have a finite life and are subjected to daily wear and tear. The viscoelastic nature of ligaments suggests they adhere to time-dependent material behavior. The ligaments will exhibit gradual deformation and recovery when they are subjected to loading and unloading. Prolonged exposure to cyclic or static loads results in residual deformation, or creep, despite self-repair capabilities of the tissues (Claude et al., 2003; Jackson et al., 2001; Solomonow et al., 2003b; Solomonow et al., 2003c; Solomonow et al., 1999; Williams et al., 2000). The majority of creep recovery occurs in the first hour of rest (Solomonow et al., 2003c), but the remaining recovery back to the original position is extremely slow (Twomey, 1988). Research indicates that full recovery of creep induced by cyclic or static lumbar flexion is unattainable within 7 hours of rest (Claude, 2003; Gedalia, 1999; Jackson, 2001; Solomonow, 2003c; Solomonow 2000).

Damage of the passive structures of the spine caused acutely or over time reduces the active stability of the spine. Research has indicated that muscle spasms, an attempt of the body to stabilize a potentially unstable spine, result from even very mild tissue damage in the passive structures of the spine (Holm et al., 2002). The neural-feedback system increases demands on the muscles to compensate for an unstable passive subsystem (Adams and Dolan 1995; Holm et al 2002). As laxity develops within the viscoelastic tissues the imbedded mechanoreceptors are desensitized resulting in decreased signaling to the central nervous system and ultimately decreased muscle activation during loading (Solomonow et al., 1999).

Previous work has shown that reduced reflexive muscle activity, microdamage and acute inflammation of the viscoelastic tissues, and the presence of initial and delayed hyperexcitability during recovery result from a single period of static or cyclic flexion in a feline model (Claude et al, 2003; Solomonow et al., 2003b; Solomonow et al., 2003c) and in humans (Solomonow et al, 2003a) regardless of the load magnitude applied. The neuromuscular disorder is transient in nature and is predicted to diminish within 2 to 3 days of exposure. However, long-term exposure to daily loading may incur chronic inflammation (Carpenter et al., 1998; Soslowsky et al, 2000) of the tissues, permanently change ligament properties (Thornton et al., 2003; Woo et al., 1999), pain, and increased muscle activity (Fisher and Chang, 1985; Sihvonen et al, 1991; Van Dieen et al, 2003).

Sbriccoli et al (2004) studied the impact a series of short static lumbar flexions followed by an equally long rest period had on the development of a CLBD in an in vivo feline model. 10 min of flexion followed by ten min of rest were repeated six times for a total of 2 h and 60 min cumulative time exposure to static flexion at loads of 20, 40 and 60N. Viscoelastic creep and reflex electromyographic activity from the multifidus muscle were monitored during the flexion-rest sessions and the following 7-h recovery period. Load magnitude was directly correlated to the development of a neuromuscular disorder. Twenty and 40N did not elicit the development of the delayed hyperexcitability component of a neuromuscular disorder, whereas 60 N did. High load magnitudes were confirmed biomechanically as a risk factor, supporting the epidemiological data.

CHAPTER 3 – OBJECTIVES

Previous studies have shown that static lumbar flexion under constant load sustained for 20 minutes resulted in a neuromuscular disorder characterized by decreased reflexive muscle activity, spasms during the flexion and immediate muscular hyperexcitability upon rest (Solomonow et al., 2003c). However, the impact of various durations of load application on the development of a neuromuscular disorder is unknown. The insight gained from studying various durations will help define risk factors of CTD and assist in the development of an optimal dose-duration ratio to limit, attenuate, or prevent the adverse effects of static load on the lumbar spine.

Three different magnitudes of loads (20N, 40N, and 6 N) will be used in order to assess the effects of load magnitude during a 30 minute static loading, 10 minute rest, and 30 minute static loading work/rest period on creep development and its recovery. Data relating to the development of the neuromuscular disorder for a schedule of 10 minute work and 10 minute rest repeated six times (for a cumulative work time of 60 min) and for the 3 loads spanning the physiological range (20, 40, 60N) is available for comparison.

The objectives of this study are to 1.) determine the behavior of reflexive muscle activity after various magnitudes of static loading for 30 minutes loading, 10 minutes rest, 30 minutes loading, then 7 hours rest 2.) assess the development of creep in the spine's viscoelastic structures 3.) develop a model for the rotation of 30-10-30 static flexion and rest and a seven hour rest period 4.) compare with 1:1 work-to-rest duration ratio to determine which can best expedite the recovery of creep.

CHAPTER 4 – METHODOLOGY

4.1 Preparation

Twenty adult cats, 1 to 2 years old with an average weight of 4.51 kg, were anesthetized with a single injection of chloralose (60 mg/kg) in a protocol approved by the Institutional Animal Care and Use Committee (IACUC). The skin overlying the lumbar spine was dissected from the thoracic level to the sacral level and allowed to retract laterally to expose the intact dorsolumbar fascia. The preparation was positioned in a rigid stainless steel frame, which allowed for the L₁ and L₇ lumbar spinous processes to be isolated through external fixation, and fixed for subsequent EMG electrode insertion. Preparations were divided into three experimental groups, each subjected to a different load: 20N (n=6) for the first group, 40N (n=7) for the second group, and 60N (n=7) for the third group. A saline soaked gauze pad was applied over the incision during the experiment to prevent the exposed tissue from drying.

4.2 Instrumentation

Six pairs of stainless steel fine wire electromyographic (EMG) electrodes, insulated except for a 1-mm exposed tip, were inserted through hypodermic needles into the multifidus muscles of L₁-L₂, L₂-L₃, L₃-L₄, L₄-L₅, L₅-L₆, and L₆-L₇ on the right side. The insertion point was 8 mm laterally from the posterior spinous processes and the interelectrode distance of each pair was 3-4 mm. A ground electrode was inserted into the gluteus muscle. Each electrode pair constituted the input to a differential amplifier with a 110 dB common mode rejection ratio, a gain capability of up to 200,000 and a band pass filter in the range of 6-500 Hz. The electromyographic responses from each channel were continuously monitored on oscilloscopes and stored in a computer at a sampling rate of 1000 Hz.

An “S” shaped stainless steel hook was inserted around the L₄-L₅ motion segment of the supraspinous ligament and connected to the vertical actuator of a Bionic 858 Material Testing

System (MTS, Inc., Minneapolis, MN). The MTS actuator applied the load using a computer controlled loading system operated in load control mode. The vertical displacement of the actuator was also monitored continuously. The displacement and the load cell outputs were sampled into the computer at 1000 Hz along with the EMG data.

In order to isolate the lumbar spine, external fixators were applied to the L₁ and L₇ posterior spinous processes as shown in Figure 9. The external fixation was intended to limit the elicited flexion to the lumbar spine and prevent interaction of thoracic and sacral-pelvic structures. However, the external fixation was not intended to prevent any motion.

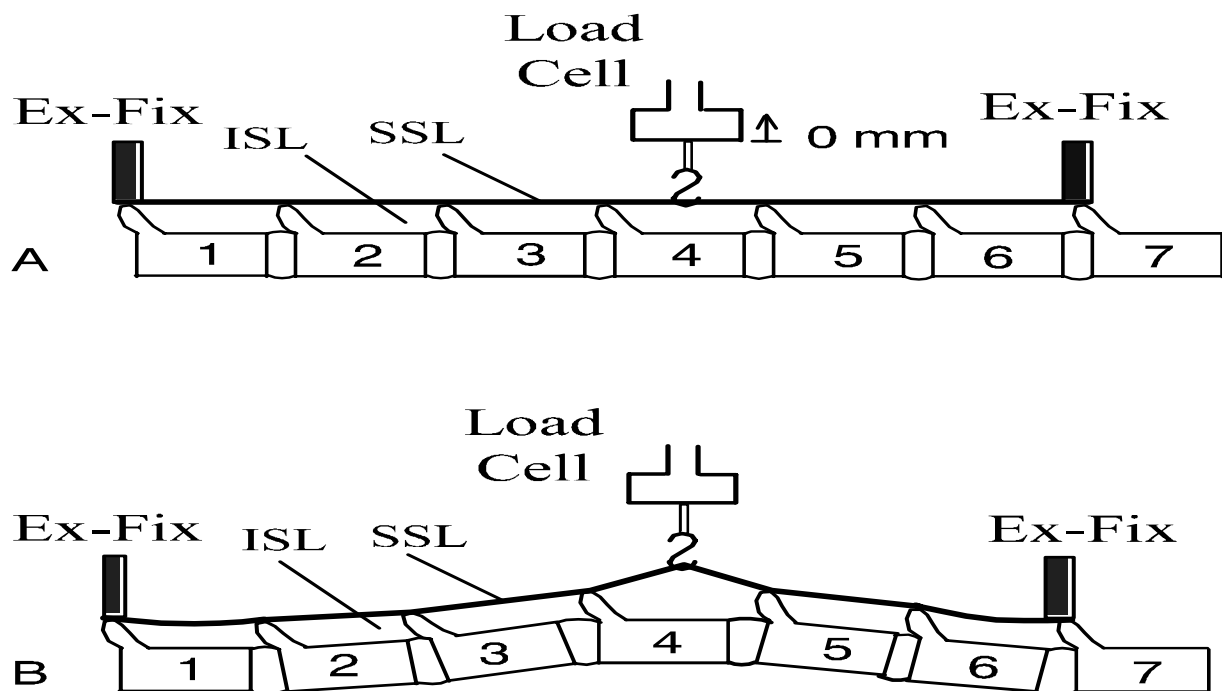


Figure 8. Schematic representation of the experimental arrangement showing the lumbar spine at rest (A), and during peak flexion (B). ISL represents the interspinous ligament and SSL is the supraspinous ligament.

4.3 Protocol

Each of the three experimental groups was subjected to the protocol detailed below. In order to standardize the initial conditions in all of the preparations, a pretension of 1N was applied to the supraspinous ligament (Eversull et al, 2001). For each experimental group, a different constant load (20, 40, or 60N) was applied to the lumbar spine via the S-shaped stainless steel hook applied to the L₄-L₅ supraspinous ligament. Lumbar flexion was created as the S-shaped hook was pulled up from a resting position by the materials testing system. The tension level remained constant during a 30 min loading period. The load was then fully removed, allowing 10 minutes in the resting position and finally, followed by a second 30 min loading period. The total exposure time to load in static flexion was 60 minutes and the total loading/rest time was 70 minutes. The EMG signal, the vertical displacement, and the load were recorded continuously during the loading periods.

During the seven hours of rest following the load/rest sessions, nine 8 second tests were performed to assess vertical displacement, associated creep and EMG recovery. Each group was loaded during the recovery with the same load used in the two 30 min tests. These tests were applied after ten minutes of rest, 30 minutes, 60 minutes and each hour thereafter. Each 8 second test was comprised of a 6 second linear increase in load followed by 2 seconds of constant load. The 8 second tests were recorded in 16 second windows triggered by the computer at the appropriate time. Between the aforementioned tests, the spine remained unloaded. Similarly, the load was increased linearly in the initial 6 seconds of the two 30 minute loading periods. Possible damage to the ligaments due to a sudden or fast stretch was avoided by linearly increasing to the desired load over a 6 second time span (Panjabi and Courtney, 2001).

Each preparation was subjected to only one load magnitude and the three load magnitudes (20, 40 and 60N) were selected to cover the range from excitation threshold (15N) to just below maximal ligament strain (70N) (Claude et al, 2003; Solomonow, 1998). The EMG, load, and supraspinous ligament displacement data were then stored in the computer for subsequent analysis.

4.4 Analysis

1.5 second windows of EMG, vertical displacement, and static load applied to the supraspinous ligament were sampled immediately at the beginning of the two 30 min loading periods and every 20 seconds thereafter for each 30 min static loading period. During the recovery period, the analysis was performed over the 2 second constant-load phase following the 6 second linear increase. In order to ensure that the load was fully applied, the first 0.5 seconds of the constant-load phase (2 second length) was discarded, and the analysis was performed over the following 1.5 seconds. Each EMG sample was full wave rectified, integrated over the 1.5 second window, and normalized with respect to the integrated EMG value obtained for the first window of the first 30 min period to compute the normalized integrated EMG (NIEMG). All corresponding NIEMG data from preparations subjected to the same load were pooled, and the mean and standard deviations were calculated and plotted on a NIEMG versus time plot for each of the muscles of the 6 lumbar levels investigated.

The displacement data were normalized to the displacement recorded at the beginning of the first 30 min loading period. The normalized displacement data of the preparations subjected to the same load were pooled and the mean and standard deviation was calculated and plotted as normalized displacement versus time.

4.5 Model

The model structure The pooled NIEMG data from each of the six lumbar levels from the multifidus muscle as well as the displacement recorded from the load cell is based on previous work by Solomonow et al in which a continuous 20 min static load was followed by a 7 h recovery period (2002; 2003c; 2003d). An exponential model was chosen because it represents the classic response of viscoelastic materials to loads or elongation. The equations were altered to describe a series of work periods spaced by rest periods by adding two new components. T_W is the time period over which work (i.e., load) was applied to the spine, a value of 30 min for the present study. T_R is the time of rest between two sequential work periods (T_W), which is 10 min in this study. Equation (1) describes the NIEMG behavior during each of the work periods.

$$NIEMG(t) = A_n e^{\frac{-(t-n(T_W+T_R))}{T_{n1}}} \Big|_{n(T_W+T_R)}^{(n+1)T_W+nT_R} + NIEMG_{0n} \quad (1)$$

where $NIEMG(t)$ is the NIEMG as a function of time t , A_n is the amplitude of the exponential component of the NIEMG (unitless), T_{n1} is the exponential decay time constant (in minutes), and $NIEMG_0$ is the steady state NIEMG amplitude (unitless).

It was assumed that A and $NIEMG_0$ change from one work period to the next and are therefore not constant throughout the work-rest session. It was also assumed that T_1 might not be the same for the two work periods.

The first transient component of the recovery equation will be dominant because this study employs only a 10 min rest and the steady-state component contribution as well as the delayed hyperexcitability term can be neglected. The equation for the rest period is as follows:

$$NIEMG(t) = \left\{ t - [(n+1)T_W + nT_R] \right\} \times B_n e^{-\frac{t - [(n+1)T_W + nT_R]}{T_{n2}}} \Bigg|_{(n+1)T_W + nT_R}^{(n+1)(T_W + T_R)} + NIEMG_{0n} \quad (2)$$

where B_n is the amplitude of the exponential component of the NIEMG during recovery, and T_{n2} is the time constant of the exponential.

The development of displacement (an indirect measure of creep in the viscoelastic tissues) during the two work periods spaced by a rest period is described by the following equation:

$$DISP(t) = [D_{0n} + D_{Ln}(1 - e^{-\frac{[t - n(T_W + T_R)]}{T_{n5}}})] \Bigg|_{n(T_W + T_R)}^{(n+1)T_W + nT_R} \quad (3)$$

where D_{0n} is the elastic component of amplitude; D_{Ln} is the viscoelastic component amplitude; and T_{n5} is the time constant of the creep during flexion. T_{n5} , D_{0n} and D_{Ln} were assumed to be variables.

The recovery of displacement during the rest period is described by:

$$DISP(t) = [D_{0n} + R_n + (D_{Ln} - R_n)e^{-\frac{t - [(n+1)T_W + nT_R]}{T_{n6}}}] \Bigg|_{(n+1)T_W + nT_R}^{(n+1)(T_W + T_R)} \quad (4)$$

where R_n is the residual creep at the end of each rest session, and T_{n6} is the time constant governing the recovery of creep in each rest session.

The long-term 7 h recovery after the work-rest-work session was modeled by the original equation for long-term recovery (Claude, 2003; Solomonow, 2003c). The model for displacement is described in eq. (5).

$$DISP(t) = D_0 + R + (D_L - R)e^{-t/T_6} \quad (5)$$

where D_0 is the elastic component amplitude of displacement (in millimeters), D_L is the viscoelastic component amplitude at the end of 70 min (in millimeters), R is the residual creep at the end of recovery (in millimeters), and T_6 is the recovery time constant (in minutes).

For the NIEMG, the model format was:

$$NIEMG(t) = E(1 - e^{-t/T_3}) + tBe^{-t/T_2} + C(t - T_d)e^{-(t-T_d)/T_4} + NIEMG_0 \quad (6)$$

where $E(1 - e^{-t/T_3})$ represents the steady-state recovery component, tBe^{-t/T_2} is a transient hyperexcitability component, and $C(t - T_d)e^{-(t-T_d)/T_4}$ the delayed transient hyperexcitability (“morning after”). This term becomes functional only for $t \geq T_d$. $NIEMG_0$ represents the residual response at the end of 20-min constant load (unitless).

In this model, the constraint of $E + NIEMG_0 = 1$ is used to ensure that full recovery results in a normal (unity) response. E , B , and C are unitless. T_4 , T_5 , T_6 , and T_d are expressed in minutes. The second and third terms, therefore, are transient features that first increase and then reverse (decrease) over time to finally arrive near zero as the effect of hyperexcitability diminishes with rest. Furthermore, the third term, which represents the delayed hyperexcitability, becomes effective only after $t \geq T_d$; that is, the effect of this term is null until recovery time exceeds T_d . Overall, the model provides a unique prediction of the NIEMG at any given time during a rest period following a static loading period.

Once the means \pm SD of the experimental data were calculated, attempts were made to generate the best-fit models described above by using Marquardt-Levenberg nonlinear regression algorithm. In some cases, the algorithm failed to converge satisfactorily; in these cases, initial or final values were determined by sequential recursive iteration, optimizing for regression coefficient.

CHAPTER 5 – RESULTS

5.1 Raw EMG

The raw EMG activity represents the direct muscle activity of each preparation while under loading. The EMG progressively decreases over time in both loading periods; however, it is more evident in the second loading period. Note the random presence of spasms during both loading periods and during the 7 h recovery. The raw EMG, load, and displacement of a preparation at 20N, 40N, and 60N are shown in Figures 10, 11 and 12, respectively.

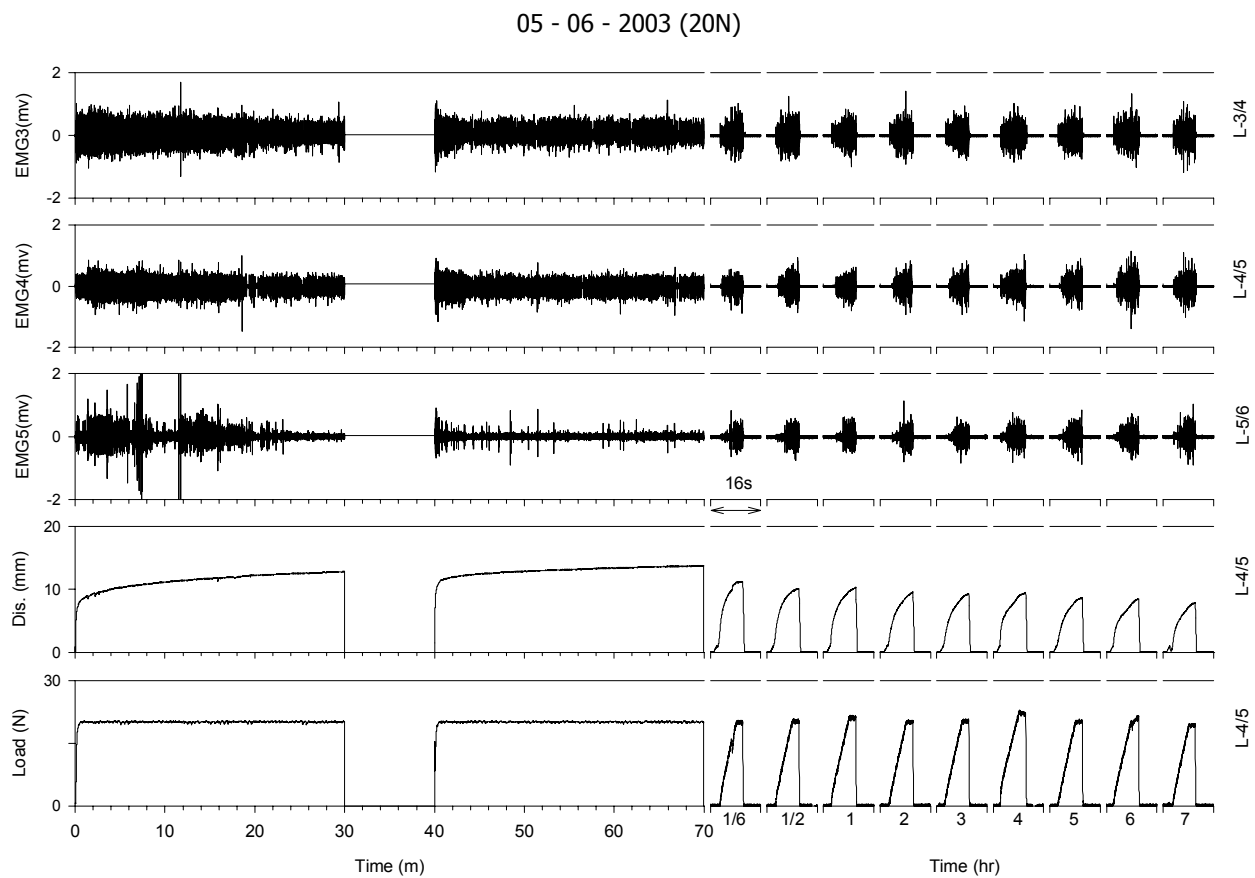


Figure 9. Typical recording of EMG from the L₃-L₄ through the L₅-L₆ multifidus and lumbar displacement and static load recorded from 1 preparation subjected to a load of 20N.

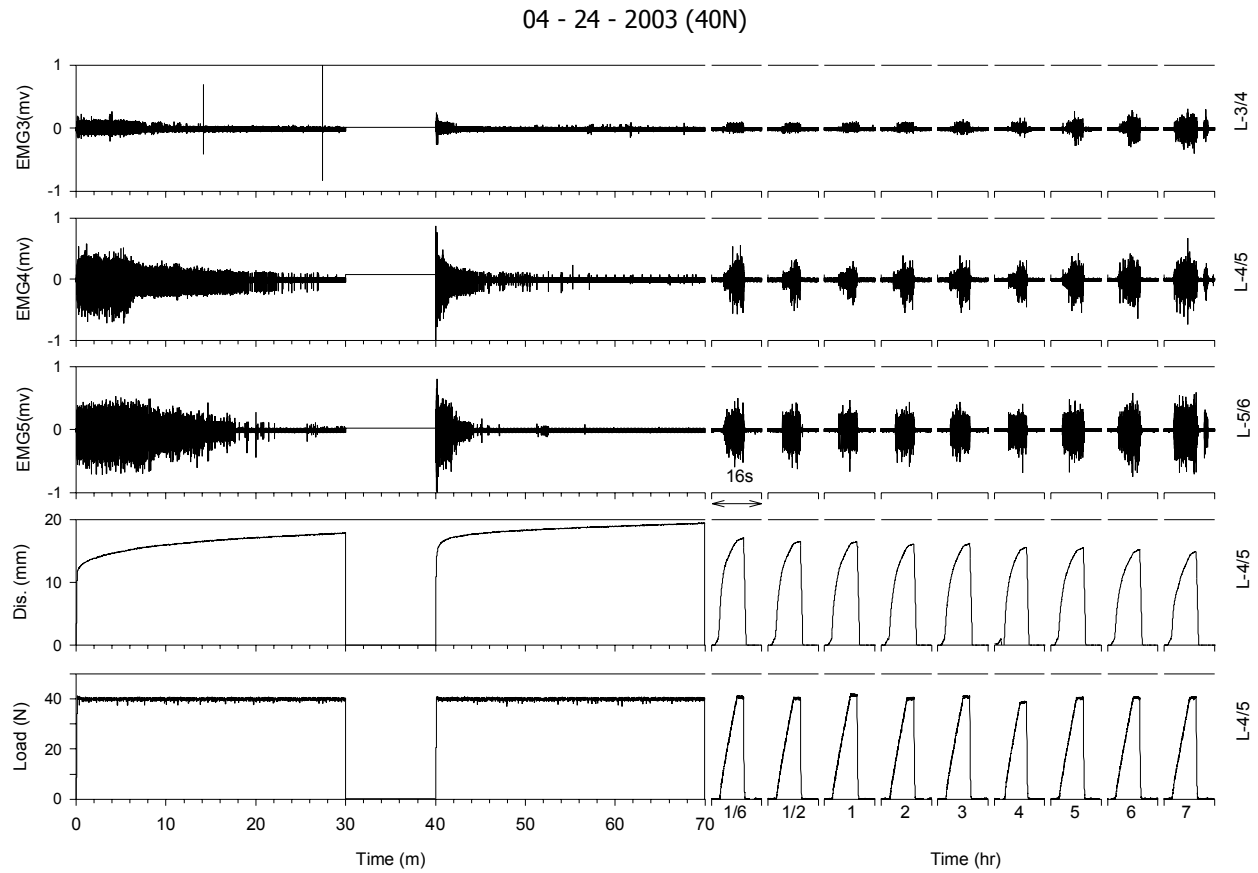


Figure 10. Typical recording of EMG from the L₃-L₄ through the L₅-L₆ multifidus and lumbar displacement and static load recorded from 1 preparation subjected to a load of 40N.

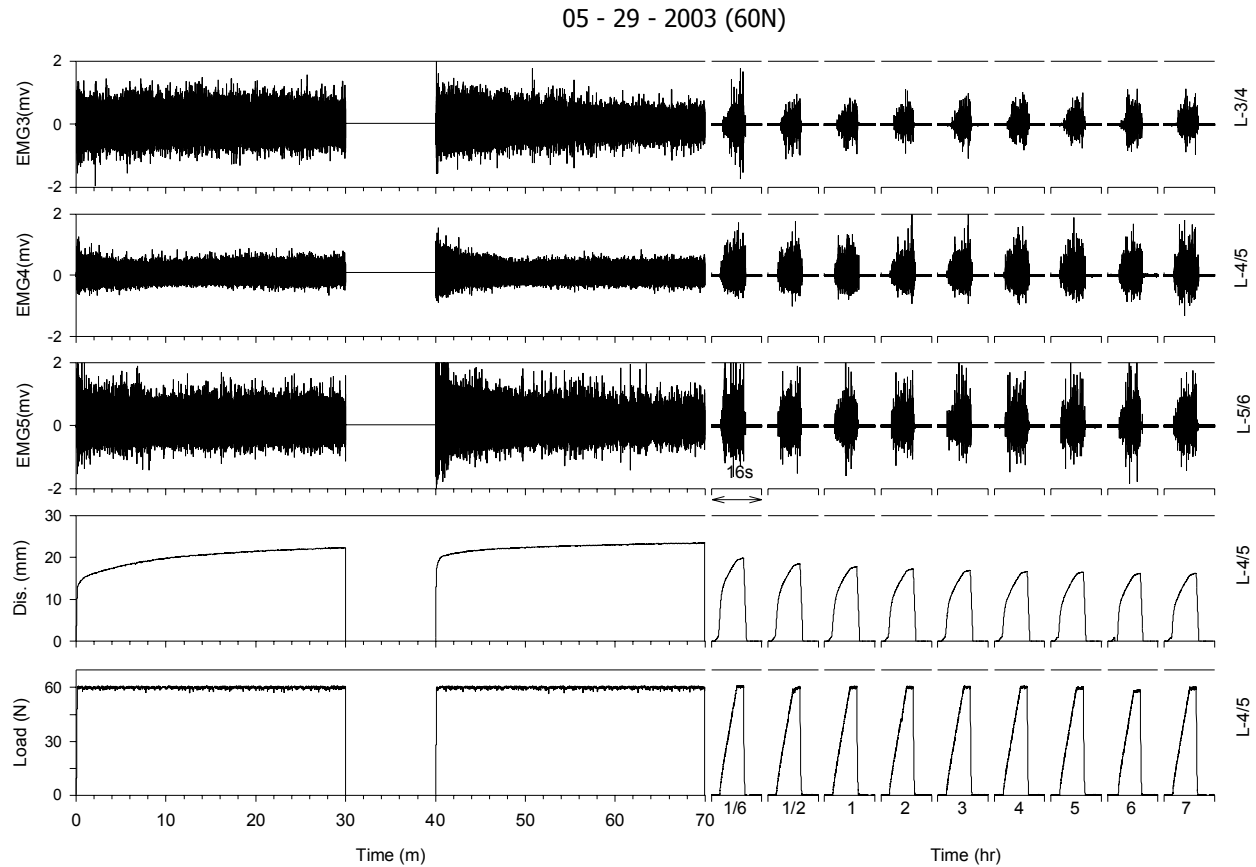


Figure 11. Typical recording of EMG from the L₃-L₄ through the L₅-L₆ multifidus and lumbar displacement and static load recorded from 1 preparation subjected to a load of 60N.

5.2 NIEMG Results

Low EMG activity within the multifidus was noticed at the L-1/2, L-2/3 and L-6/7 lumbar levels throughout loading and recovery. Most likely, this is the result of the increased distance from the load point (L₄-L₅). Therefore, only the recordings and data for L₃-L₄, L₄-L₅, and L₅-L₆ multifidus will be examined.

5.2.1 20N Loading

The mean NIEMG in the L₃-L₄, L₄-L₅, and L₅-L₆ multifidus subjected to 20N load decreased from 1.0 to 0.5180 (48.2% decrease), 0.3782 (62.2% decrease), and 0.3717 (62.8% decrease), respectively, during the initial 30 min loading session. The mean NIEMG recovered to 0.7631, 0.6774 and 0.6963 respectively, during the 10 min rest period. At the end of the 2nd 30

min loading period, the mean NIEMG decrease for each of the six lumbar levels was 56.1%, 75.4%, and 66.2%, respectively.

During the 7 hour recovery period, the NIEMG exhibited an increase in the first 10 minutes followed by a minor decrease for approximately 2 hours and then an extended increase to 151%, 137%, and 164% of the initial values at the end of rest, in the L₃-L₄, L₄-L₅, and L₅-L₆ multifidus muscles, respectively. Table 1 provides the mean NIEMG values for the beginning and end of each loading period and for the 7 hour recovery period for the preparations exposed to 20N static loading. The mean NIEMG values for both flexion periods and the corresponding 7 hour recovery for the three lumbar levels and the displacement of the group exposed to 20N are shown in Figure 13.

Table 1. Mean NIEMG Values for the group subjected to 20N Static Loading

Time (min)	L ₃ -L ₄	L ₄ -L ₅	L ₅ -L ₆
0	1.0	1.0	1.0
30	0.5180	0.3782	0.3717
40	0.7631	0.6774	0.6963
70	0.4391	0.2458	0.3385
80	0.8978	0.7628	0.7808
100	0.8669	0.7741	0.8074
130	0.8053	0.7244	0.7143
190	0.9237	0.7779	0.7715
250	0.8787	0.8173	0.8153
310	1.2160	1.0418	1.0893
370	1.4366	1.2212	1.2563
430	1.4731	1.3169	1.4710
490	1.5089	1.3715	1.6382

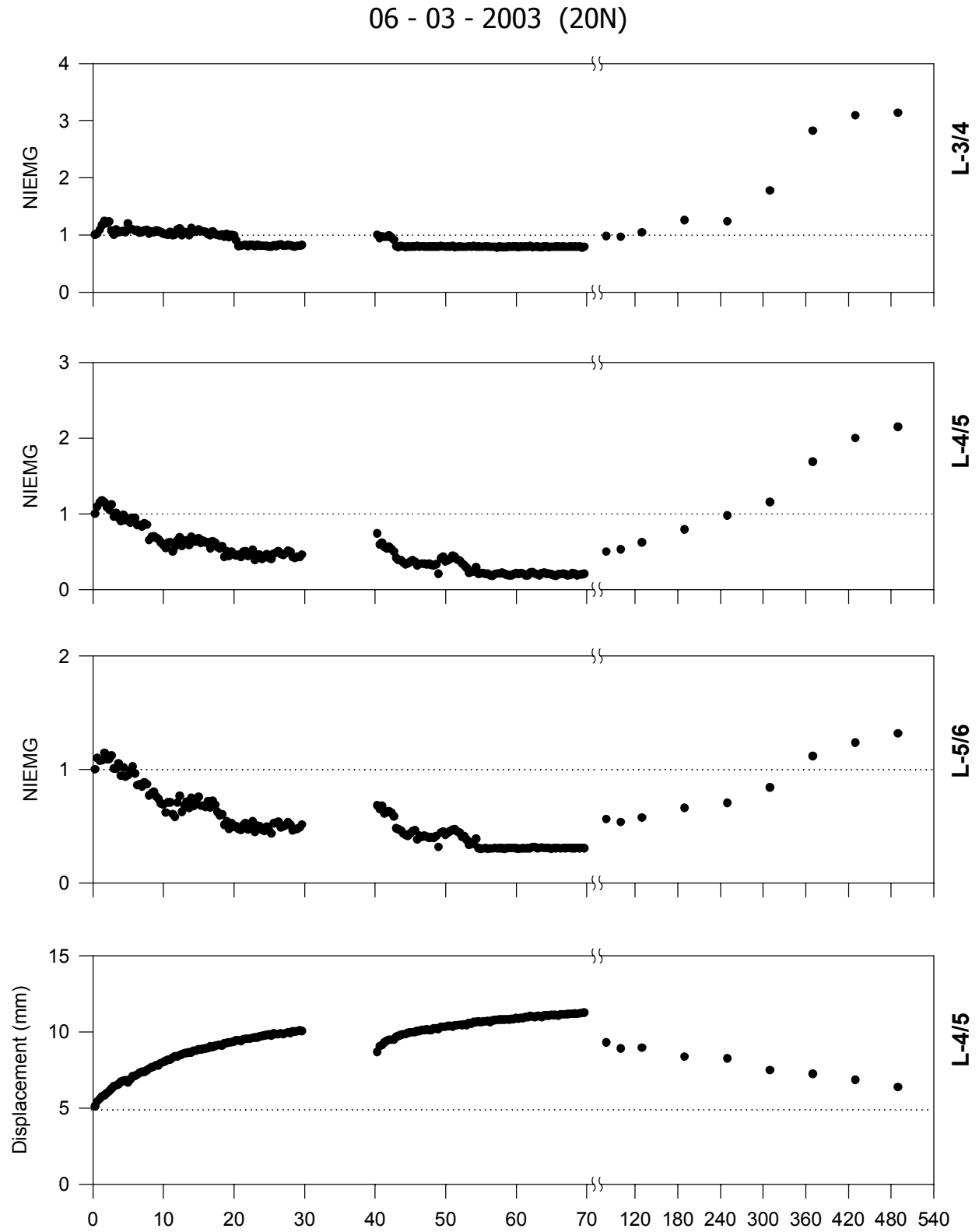


Figure 12. Mean NIEMG for L₃-L₄, L₄-L₅, and L₅-L₆ and lumbar spine displacement for 20N load.

5.2.2 40N Loading

For flexion loads of 40N, the NIEMG showed a mean decrease of 64.3%, 69.2%, and 68.3% of the initial NIEMG values at L₃-L₄, L₄-L₅, and L₅-L₆, respectively, during the first 30 min flexion session. After an intermittent 10 min rest, the levels recovered partially, exhibiting only a 27 – 37% decrease in NIEMG values. After the final 30 min flexion period, NIEMG for each of the respective levels was 69%, 75%, and 55% of the original values. During the 7 hour recovery period, the mean NIEMG followed the same pattern as 20N, gradually increasing to the final NIEMG values of 154%, 138%, and 164% of the initial NIEMG values at the end of 7 hours.

Table 2. Mean NIEMG Values for the group subjected to 40N Static Loading

Time (min)	L ₃ -L ₄	L ₄ -L ₅	L ₅ -L ₆
0	1.0	1.0	1.0
30	0.3587	0.3085	0.3169
40	0.7298	0.7311	0.7129
70	0.3075	0.2456	0.2665
80	0.8236	0.8762	0.9157
100	0.7692	0.7728	0.8194
130	0.6981	0.6693	0.6891
190	0.7412	0.7659	0.8026
250	0.8932	0.8948	0.8958
310	1.0348	1.0344	1.0509
370	1.2587	1.1966	1.3212
430	1.4160	1.2799	1.4751
490	1.5417	1.3752	1.6432

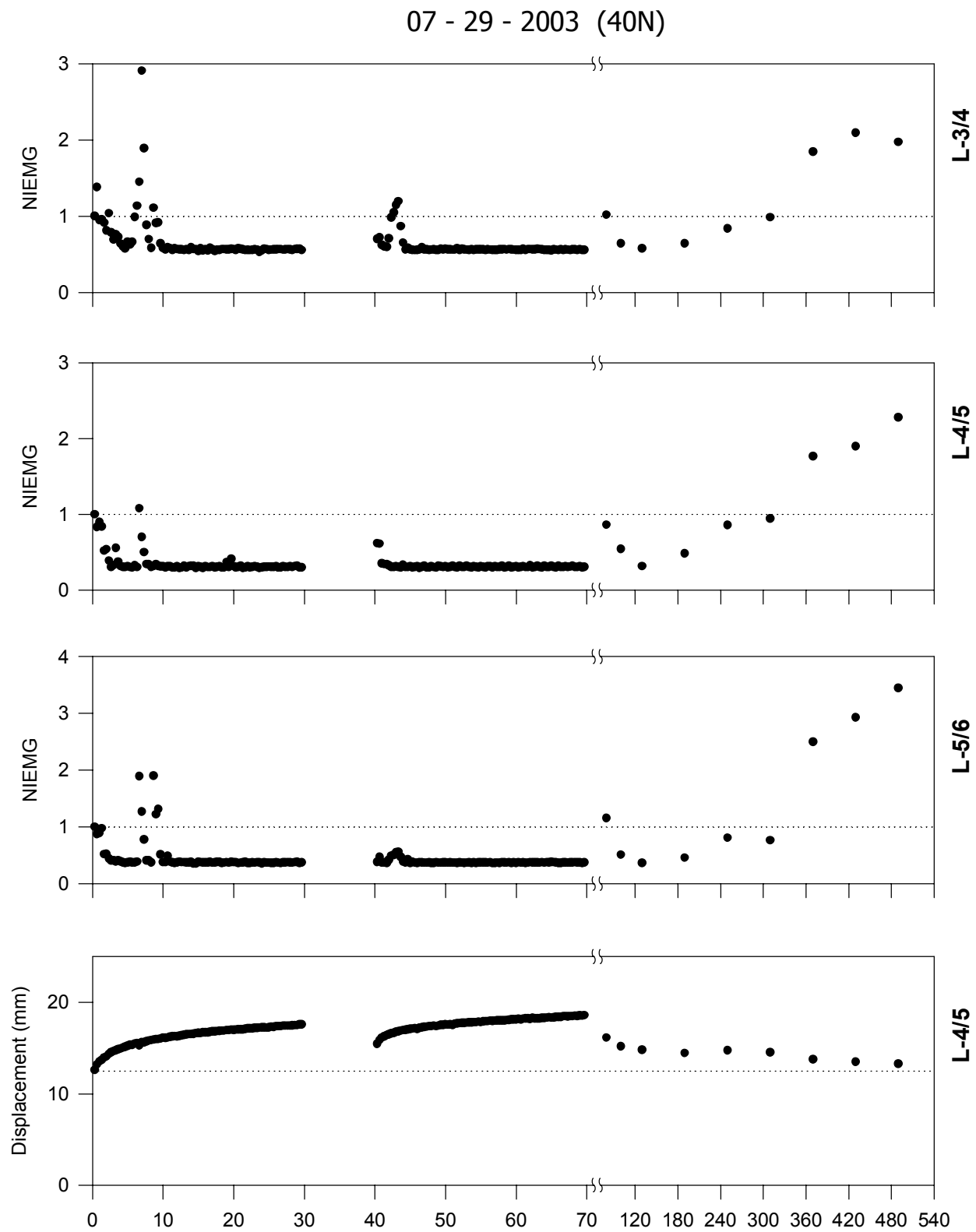


Figure 13. Mean NIEMG for L₃-L₄, L₄-L₅, and L₅-L₆ and lumbar spine displacement for 40N load.

5.2.3 60N Loading

Similarly, for 60N flexion load, reduction in NIEMG to 74%, 82%, and 66% were observed at the end the first 30 min flexion session, followed by recovery to only a 19%, 7%, and 16% decrease in NIEMG at the end of the 10 min rest period. The final mean NIEMG was a 77, 83, and 69 percent decrease of initial values at the end of the 2nd 30 min flexion period. During the recovery period, the mean NIEMG peaked within the first 10 min, decreased somewhat and then gradually increased to 113%, 124%, and 157% of the initial values at the end of the 7 hours. It is important to note that the 1.0 preload value was reached within the first 10 min of recovery and although this decreased over the first two hours, it was again reached by the second hour of recovery in L₄-L₅ and L₅-L₆. This is significantly earlier than observed in the 20 and 40N load groups (i.e., preload was exceeded in the 4th hour).

Table 3. Mean NIEMG Values for the group subjected to 60N Static Loading

Time (min)	L ₃ -L ₄	L ₄ -L ₅	L ₅ -L ₆
0	1.0	1.0	1.0
30	0.2587	0.1785	0.3381
40	0.8073	0.9350	0.8454
70	0.2318	0.1692	0.3127
80	0.9512	1.2173	1.1020
100	0.7953	1.0874	1.0259
130	0.6782	0.8974	0.8226
190	0.7636	1.0009	1.0159
250	0.8505	1.0698	1.1352
310	0.9857	1.1588	1.2848
370	1.0752	1.1421	1.3428
430	1.1704	1.1879	1.4588
490	1.1310	1.2360	1.5656

07 - 24 - 2003 (60N)

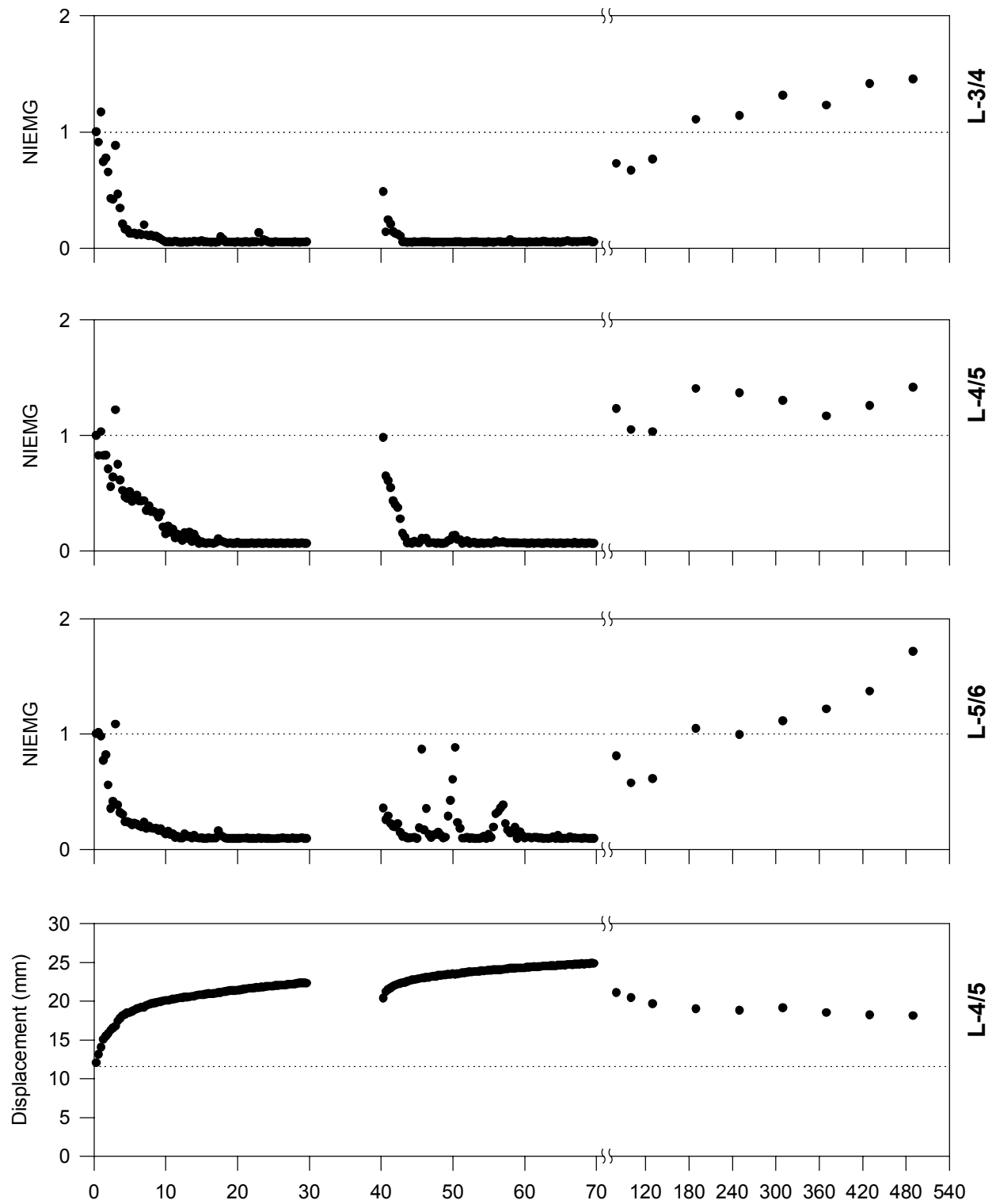


Figure 14. Mean NIEMG for L₃-L₄, L₄-L₅, and L₅-L₆ and lumbar spine displacement for 60N load.

5.3 Lumbar Spine Displacement

The vertical displacement of the lumbar spine provides an indirect measure of the overall creep developed in the supraspinous ligament of the spine during static loading and recovery.

Table 4 provides the initial mean vertical displacement, mean displacement at the end of the 1st and 2nd 30 min flexions and after the 10 min rest, and at the 7th hour of recovery for each load intensity.

Table 4. Mean displacement of the lumbar spine during loading and recovery.

Load (N)	Mean Initial Displacement (mm)	Displacement at end of 1 st 30-min session (mm)	Displacement at end of 10-min rest (mm)	Displacement at end of 2 nd 30-min session (mm)	Displacement at end of 7 hr recovery (mm)
20	5.96	11.81	10.30	12.73	8.0441
40	10.77	17.93	15.28	19.05	11.6415
60	13.01	20.52	18.27	22.07	16.16

The mean initial displacement in the preparations subjected to 20N load was 5.96 mm, which increased to 11.8089 mm, indicating a mean creep of 98.12%. During the following 10 min rest period it recovered to 10.2967 mm or 72.8% residual creep. In the second 30 min flexion the mean residual creep accumulated, resulting in a mean cumulative creep value of 113.53%. The mean cumulative creep decreased to 34.96% following the 7 hour recovery period.

For preparations exposed to a 40N flexion load, ligamentous displacement increased to 17.9295 mm at the end of the first 30 min from the mean initial displacement of 10.7651 mm, accumulating a mean creep of 66.55%. The rest period allowed the mean creep to recover to 41.98%. The mean displacement reached 11.6415 mm, a 76.98% residual creep, which improved to 8.41% during recovery.

Under a 60N load, a creep of 57.65% occurred at the end of the initial 30 min flexion period and accumulated to a final mean cumulative creep of 69.67% at the end of the second

loading period and recovered to a mean residual creep of 24.16% at the end of the 7 hour rest period.

5.4 Statistical Analysis

A two-way ANOVA was performed to determine the effect of time and load magnitude on the NIEMG and displacement data for L₃-L₄, L₄-L₅, and L₅-L₆. We are interested in determining whether applied load or time is statistically significant factors for the displacement and NIEMG changes. The PROC GLM procedure within SAS was used. Load and time are fixed effects and NIEMG or displacement of each preparation is the random effect parameter, meaning the variability of the data is affected by unknown random variables. We tested the influence of load, time, and the Load*time interaction had on the NIEMG and displacement values. Significance was set at 0.05 for all statistical tests. A summary of the results for the 1st and 2nd 30 min loading periods are shown in Table 5 and Table 6, respectively. Table 7 contains the ANOVA results for L₃-L₄, L₄-L₅, and L₅-L₆ during the 7 hour recovery period. The effects of load, time and the load*time interaction are presented in Table 8.

Table 5. F values and Pr>F values obtained for the 1st 30 min loading period for L₃-L₄, L₄-L₅, and L₅-L₆.

Tested Effects	L ₃ -L ₄		L ₄ -L ₅		L ₅ -L ₆	
	F	Pr>F	F	Pr>F	F	Pr>F
Load	13.74	<0.0001	38.24	<0.0001	1.43	0.2408
Time	4.43	<0.0001	22.84	<0.0001	8.69	<0.0001
Load*Time	0.51	0.9991	0.28	1.0000	0.19	1.0000

Table 6. F values and Pr>F values obtained for the 2nd 30 min loading period for L₃-L₄, L₄-L₅, and L₅-L₆.

Tested Effects	L ₃ -L ₄		L ₄ -L ₅		L ₅ -L ₆	
	F	Pr>F	F	Pr>F	F	Pr>F
Load	30.96	<0.0001	7.56	0.0006	5.21	0.0058
Time	3.23	<0.0001	10.27	<0.0001	1.73	0.0098
Load*Time	0.21	1.0000	0.52	0.9990	0.56	0.9969

Table 7. F values and Pr>F values obtained for the 7 hour recovery period for all six lumbar levels.

Tested Effects	L ₃ -L ₄		L ₄ -L ₅		L ₅ -L ₆	
	F	Pr>F	F	Pr>F	F	Pr>F
Load	1.85	0.1594	1.47	0.2317	1.07	0.3441
Time	7.12	<0.0001	11.32	<0.0001	8.27	<0.0001
Load*Time	0.19	0.9999	0.49	0.9581	0.12	1.0000

Table 8. F values and Pr>F values obtained for both loading periods and the 7 hour recovery period for the vertical displacement in the L₄-L₅ supraspinous ligament.

Tested Effects	1 st 30-min		2 nd 30 min		7-hour recovery	
	F-value	Pr>F	F-value	Pr>F	F-value	Pr>F
Load	655.97	<0.0001	1150.98	<0.0001	157.26	<0.0001
Time	9.68	<0.0001	3.07	<0.0001	19.47	<0.0001
Load*Time	0.50	0.9993	0.05	1.0000	0.29	0.9772

Statistical analysis indicated a significant effect of time on all parameters assessed encompassing NIEMG L₃-L₄, NIEMG L₄-L₅, NIEMG L₅-L₆ and displacement for both of the loading periods and the recovery period, showing that the NIEMG and displacement parameters are changing as time progresses. There was no significant difference between 20N, 40N, and 60N for L₃-L₄, L₄-L₅, and L₅-L₆ during recovery, implying that load magnitude is not the main determinant in the development of the disorder. However, there is a significant difference between loads during the loading periods. There was no significant differences in the load*time interaction.

5.5 Model Development

Modeling of the mean NIEMG and displacement data was performed on the lumbar levels of the most interest (L_3-L_4 , L_4-L_5 , and L_5-L_6). The best-fit model constants developed for the displacement and NIEMG data are superimposed on the experimental data as shown in Figures 16, 17, and 18 for 20, 40 and 60N respectively.

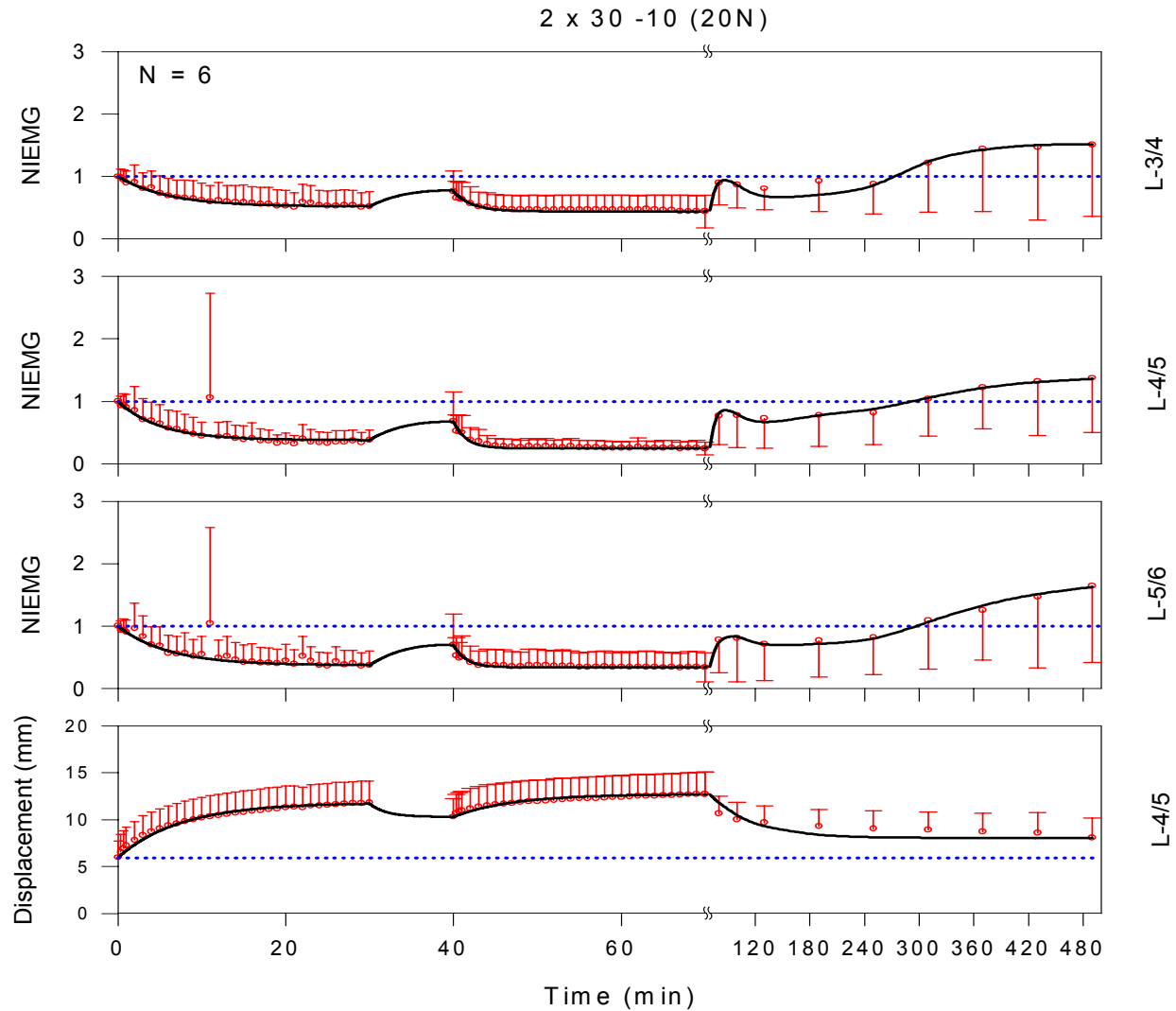


Figure 15. Mean (± SD) of the L₃-L₄, L₄-L₅, and L₅-L₆ multifidus and mean displacement during the 30:10:30 min session and 7 h recovery for a 20N load.

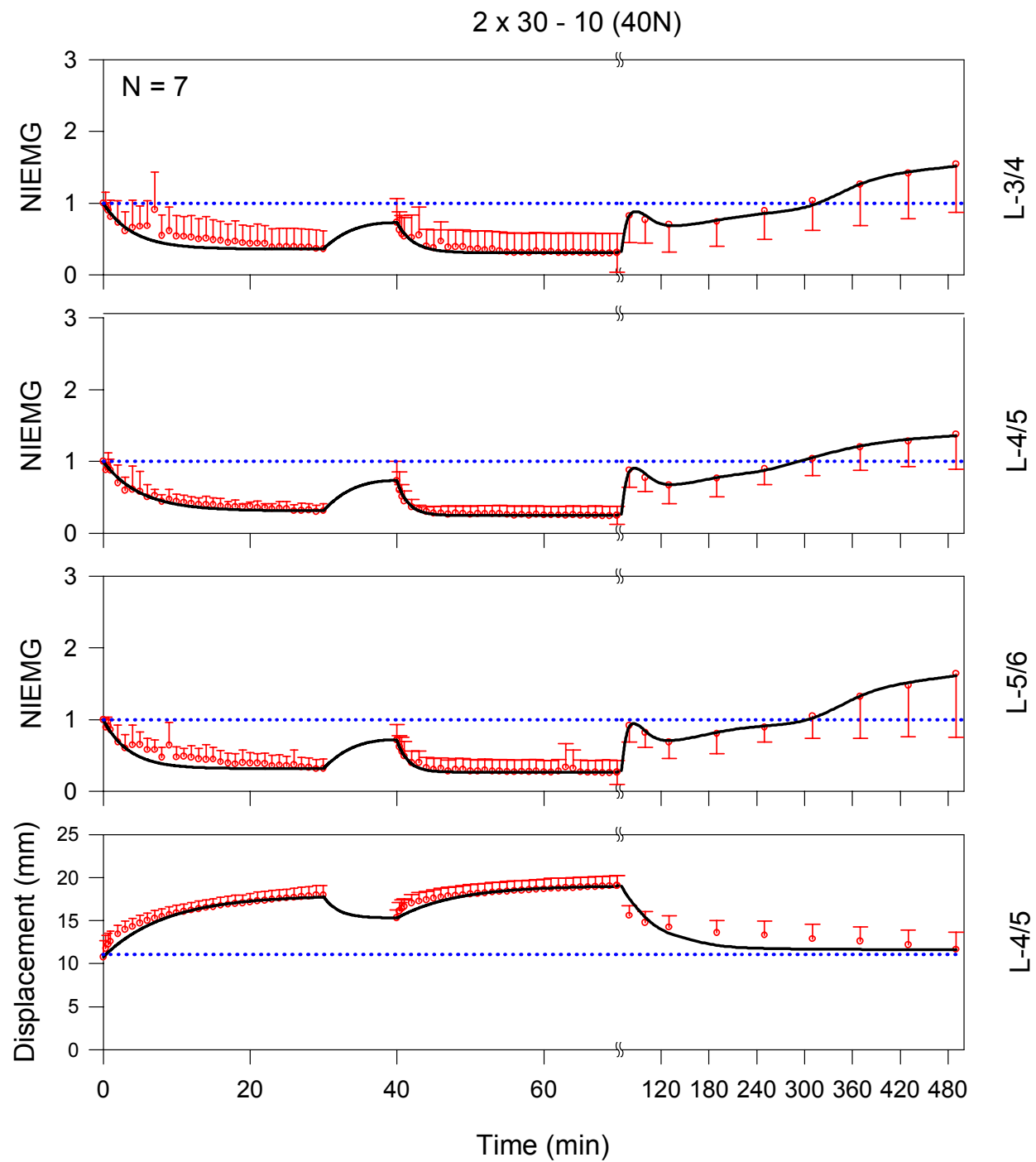


Figure 16. Mean (\pm SD) of the L₃-L₄, L₄-L₅, and L₅-L₆ multifidus and mean displacement during the 30:10:30 min session and 7 h recovery for a 40N load.

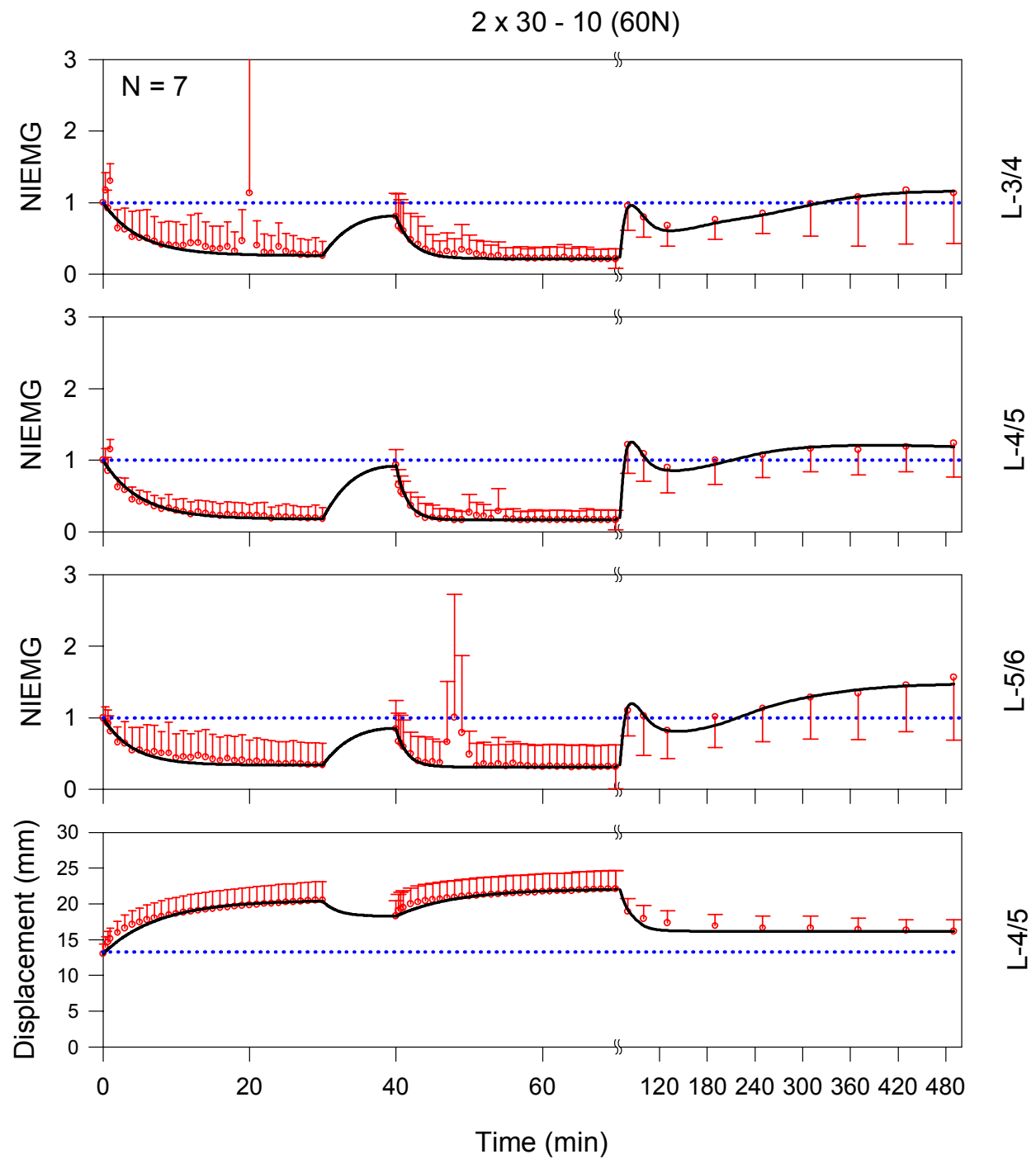


Figure 17. Mean (\pm SD) of the L₃-L₄, L₄-L₅, and L₅-L₆ multifidus and mean displacement during the 30:10:30 min session and 7 h recovery for a 60N load.

5.5.1 NIEMG and Displacement Model Parameters

The parameters of the NIEMG model for the two 30 min loading periods and the 10 min rest period for the each of the loading groups, as defined by equations (1) and (2), are presented in Table 9 and Table 10. The time constant T_{n1} and constant A_n decrease between the two work periods in the L_3 - L_4 , L_4 - L_5 , and L_5 - L_6 lumbar levels. $NIEMG_{0n}$ is considered unchanged due to lack of a trend between the two working periods. The physiological data was accurately described by the models for each of the three loads with the r^2 values ranging between .841 and .981.

Table 9. NIEMG parameters for the two 30 min loading periods

Load	Level	n	A_n	T_{n1} , min	$NIEMG_{0n}$	r^2
20N	L_3 - L_4	0	0.479	6	0.521	0.974
		1	0.321	2.2	0.442	0.872
	L_4 - L_5	0	0.620	5	0.380	0.758
		1	0.430	1.5	0.247	0.938
	L_5 - L_6	0	0.624	6	0.376	0.736
		1	0.357	1.5	0.339	0.872
40N	L_3 - L_4	0	0.641	4	0.359	0.581
		1	0.419	2.2	0.311	0.814
	L_4 - L_5	0	0.689	5	0.311	0.958
		1	0.484	2.2	0.247	0.971
	L_5 - L_6	0	0.683	5	0.317	0.715
		1	0.445	1.5	0.268	0.945
60N	L_3 - L_4	0	0.741	4	0.259	0.511
		1	0.585	2.2	0.222	0.935
	L_4 - L_5	0	0.822	3.7	0.178	0.919
		1	0.770	1.2	0.165	0.948
	L_5 - L_6	0	0.662	3.5	0.338	0.884
		1	0.531	1.5	0.314	0.175

Table 10. NIEMG parameters for the intermittent 10 min rest period

Load	Level	n	NIEMG _{0n}	B _n	T _{n2}
20N	L ₃ -L ₄	0	0.519	0.07	10
	L ₄ -L ₅	0	0.380	0.08	10
	L ₅ -L ₆	0	0.372	0.09	10
40N	L ₃ -L ₄	0	0.361	0.1	10
	L ₄ -L ₅	0	0.311	0.115	10
	L ₅ -L ₆	0	0.317	0.11	10
60N	L ₃ -L ₄	0	0.261	0.15	10
	L ₄ -L ₅	0	0.180	0.2	10
	L ₅ -L ₆	0	0.338	0.14	10

The displacement model given in equation (3) and (4) was fitted to the mean data collected during the two 30 min static flexion loading periods. In the displacement model, we observe that D_{0n} increases and D_{Ln} decreases from the first to the second loading session and the time constant T_{n5} remains relatively constant. Table 11 and Table 12 contain the model parameters for displacement.

Table 11. Displacement parameters for the two 30 min loading periods

Load	n	D _{0n} , mm	D _{Ln} , mm	T _{n5} , min	r ²
20N	0	5.960	4.307	7.5	0.981
	1	10.297	1.848	7	0.920
40N	0	10.765	4.955	8.5	0.943
	1	15.284	2.688	8	0.841
60N	0	13.014	5.525	7.5	0.939
	1	18.269	2.720	8	0.870

Table 12. Displacement parameters for the intermittent 10 min rest period

Load	n	D _{0n} , mm	D _{Ln} , mm	R _n , mm	T _{n6} , min
20N	0	5.960	4.307	4.337	2
40N	0	10.765	4.955	4.519	2
60N	0	13.014	5.525	5.255	2

The recovery behavior of NIEMG is described by equation (6). The constants determined by the analysis are given in Table 13. R² values ranged from .942 to .996 indicating a good fit

between the model and physiological data. There is an indication of variations between the loading groups.

Table 13. NIEMG Model Parameters during 7 hour recovery

Load	Level	E	T ₃ , min	B	T ₂ , min	C	T ₄ , min	T _d , min	NIEM G ₀	r ²
20N	L ₃ -L ₄	0.561	190	0.085	15	0.008	200	240	0.439	0.942
	L ₄ -L ₅	0.754	100	0.1	14	0.0035	300	270	0.246	0.991
	L ₅ -L ₆	0.661	150	0.06	19	0.0055	370	270	0.339	0.990
40N	L ₃ -L ₄	0.693	115	0.09	15	0.006	250	300	0.307	0.991
	L ₄ -L ₅	0.754	100	0.11	14	0.0035	300	270	0.246	0.994
	L ₅ -L ₆	0.733	85	0.12	13	0.0065	275	300	0.267	0.996
60N	L ₃ -L ₄	0.784	120	0.14	13	0.0025	200	275	0.216	0.984
	L ₄ -L ₅	0.835	45	0.2	12	0.003	190	200	0.165	0.985
	L ₅ -L ₆	0.687	64	0.16	13	0.004	320	200	0.313	0.974

The constants associated with the vertical displacement model for the 7 h recovery period as described in equation (5) are presented in Table 14. The model fits well with the physiological data with r² ranging from .705 to .892.

Table 14. Displacement Model Parameter during 7 hour Recovery

Load	D ₀ , mm	D _L , mm	R, mm	T ₆ , min	r ²
20N	5.960	6.797	2.084	46	0.705
40N	10.765	8.287	0.877	44	0.719
60N	13.014	9.067	3.144	15	0.892

CHAPTER 6 – DISCUSSION and CONCLUSION

Epidemiological data and scientific research have continuously validated the theory that static and cyclic loadings are primary risk factors in the development of neuromuscular disorders (Claude et al., 2003; Hoogendoorn et al., 2000; Punnett et al., 1991; Silverstein et al., 1986; Solomonow et al., 2003c; Solomonow et al., 2003d). Currently, it is of primary interest to examine the effects and work organization factors, such as work-rest cycles, in order for effective intervention strategies to be designed and implemented. This study demonstrates that feline preparations exposed to longer periods (two 30-min sessions) of static flexion spaced by a 10 min interval (a 3:1 work-rest duration ratio) developed a neuromuscular disorder whereas six 10 minute sessions with identical rest duration did not.

The neuromuscular disorder was characterized by the random and unpredictable presence of spasms, an indication of the existence of tissue microdamage, and decreased reflexive muscle activity during the flexion periods. Initial and delayed hyperexcitability appeared in all of the preparations regardless of load magnitude. Not only was full recovery not observed in any of the preparations but also at the end of a 7-hr recovery period, the NIEMG of the L₁-L₂, L₂-L₃, L₃-L₄, L₄-L₅, L₅-L₆, and L₆-L₇ for 20, 40 and 60N was near or above the 1.0 initial value, indicating the development of a severe neuromuscular disorder.

Multiple factors such as high magnitude and increasing number of repetitions have been biomechanically proven to cause the development of neuromuscular disorders of the lumbar spine (Sbriccoli et al, 2004; Sbriccoli et al, In Press). Although differences were evident between the three groups subjected to differing magnitudes of load in the model components, they were not statistically significant. This is compelling evidence that the duration of the work period, not load magnitude, was the prominent factor in promoting the disorder.

During the first 30 min period of loading the NIEMG decreased significantly, partially recovered during the 10 min rest and exceeded the initial NIEMG decrease after experiencing a second exposure to 30 minutes of load. At the end of loading, NIEMG had decreased to 21-75% of its preload values for 20N, 35-75% for 40N, and 45-83% for 60N. A slight trend of larger increase in NIEMG decrease for larger loads emerged indicating a greater injury potential at higher loads. Interestingly, model data confirmed a correlation between larger NIEMG decay in tissues exposed to larger loads. The time constant T_{n1} notably decreased between the loads in the first loading period. T_{n1} ranged from 5-6 min for 20N, 5-4 min for 40N, and 3.5-4 min for 60N. However, at the end both loading sessions a marked decrease was evident between the two 30 min loading periods. The T_{n1} values had decreased from an average of 5.67 min, 4.67, and 3.73 min in the first loading period to a mean average of 1.73 min, 1.96 min, and 1.63 min after both loading periods in 20, 40 and 60N respectively, indicating the EMG decreases faster as time progresses. Interestingly, T_{n1} remained nearly constant for the three different loads during the second 30 min loading session, but decreased as the load increased in the first 30 min loading session. This confirms that the effect of load diminishes as the loading time increases, making a longer work period the major factor in the development of a neuromuscular disorder.

The second component of a neuromuscular disorder, spasms, was also present. Duration, amplitude, location, and timing of the spasms were unpredictable and erratic. Spasms are an attempt by the musculature of the spine to maintain joint stability as the pain afferents in the microdamaged collagenous structures of the viscoelastic tissues are eliciting the reflex activation of the muscles. Spasms modify the mean values of NIEMG and increase the standard deviation sometimes causing an artificially low r^2 value in the models.

A peak in the NIEMG was observed in all of the experimental groups during the first hour of recovery, an attempt through increased activation of the muscles to protect the strained viscoelastic tissues from further injuries. This is recognized as the initial hyperexcitability period of recovery. The hyperexcitability peaked within the first ten minutes of recovery for 40 and 60N. The hyperexcitability peaked slightly slower in the group exposed to 20N peaking between 30 min and 2 hours into recovery. The mean values obtained for the peak of the initial hyperexcitability were close to the 1.0 preload value (0.90, 0.82, and 0.90 for the 20, 40, and the 60N groups, respectively), and when only considering the L₃-L₄, L₄-L₅, and L₅-L₆ lumbar levels the peaked values increased slightly as loads increased and the 60N group was higher than the 1.0 preload value (0.84, 0.87, and 1.09 for the 20, 40, and 60N groups, respectively). No significant difference was observed between the three different loads (20, 40 and 60N) during initial hyperexcitability.

Larger loads resulted in larger magnitude of the initial hyperexcitability as confirmed by the models. The time constant T_2 decreased from a mean value of 16 min to mean values of 14 and 12.66 min as the loads increased from 20 to 40 and 60N, respectively. Shorter time constants signify accelerated development of initial hyperexcitability. B, the constant that governs the amplitude of the initial hyperexcitability increased from a mean value of 0.08 for 20N to mean values of 0.11 and 0.17 in the preparations subjected to 40 and 60N. The increased magnitude of initial hyperexcitability for higher loads indicated a greater degree of laxity and microdamage of the collagenous structures within the viscoelastic tissues and the resultant increase in muscular compensation needed to provide stiffness to the intervertebral joints and limit further damage to the viscoelastic structures.

Following the peak NIEMG values during initial hyperexcitability, a recovery of NIEMG occurred gradually over the first two hours of rest. The manifestation of delayed hyperexcitability, the muscular compensation for the development of acute inflammation in the viscoelastic tissues, was observed next. It is evident by observing Figures 16, 17, and 18 that delayed hyperexcitability is a prominent component in 20N, 40N, and 60N. The NIEMG gradually increased and reached or nearly reached the 1.0 initial value after 4 hours of recovery in the groups subjected to 20 and 40N. For 60N, L₄-L₅ and L₅-L₆ reached the 1.0 preload value after 2 hours of recovery, and lumbar levels L₂-L₃, L₃-L₄, and L₆-L₇ essentially reached the 1.0 initial value 4 hours into the recovery phase. The modeling variables T₄ and C for delayed hyperexcitability decreased as load increased. The time constant T₄ ranged from a mean value of 290 min for 20N to the mean values of 275 and 237 min for the 40 and 60N groups, respectively. The amplitude constant, C, decreased from 0.0057 for 20N to 0.0053 and 0.0032 for 40 and 60N. This indicates that the delayed hyperexcitability occurred faster and was greater in amplitude in preparations exposed to higher loads, although the differences were small. The time constant T_d increased from 260 min to 290 min from a 20N load exposure to a 40N load exposure and decreased to 225 min for groups subjected to 60N. The tendency toward an earlier and faster response was not supported by the statistical analysis. The differences between the 20, 40 and 60N loads for the 7 hour recovery period are available in Figure 19.

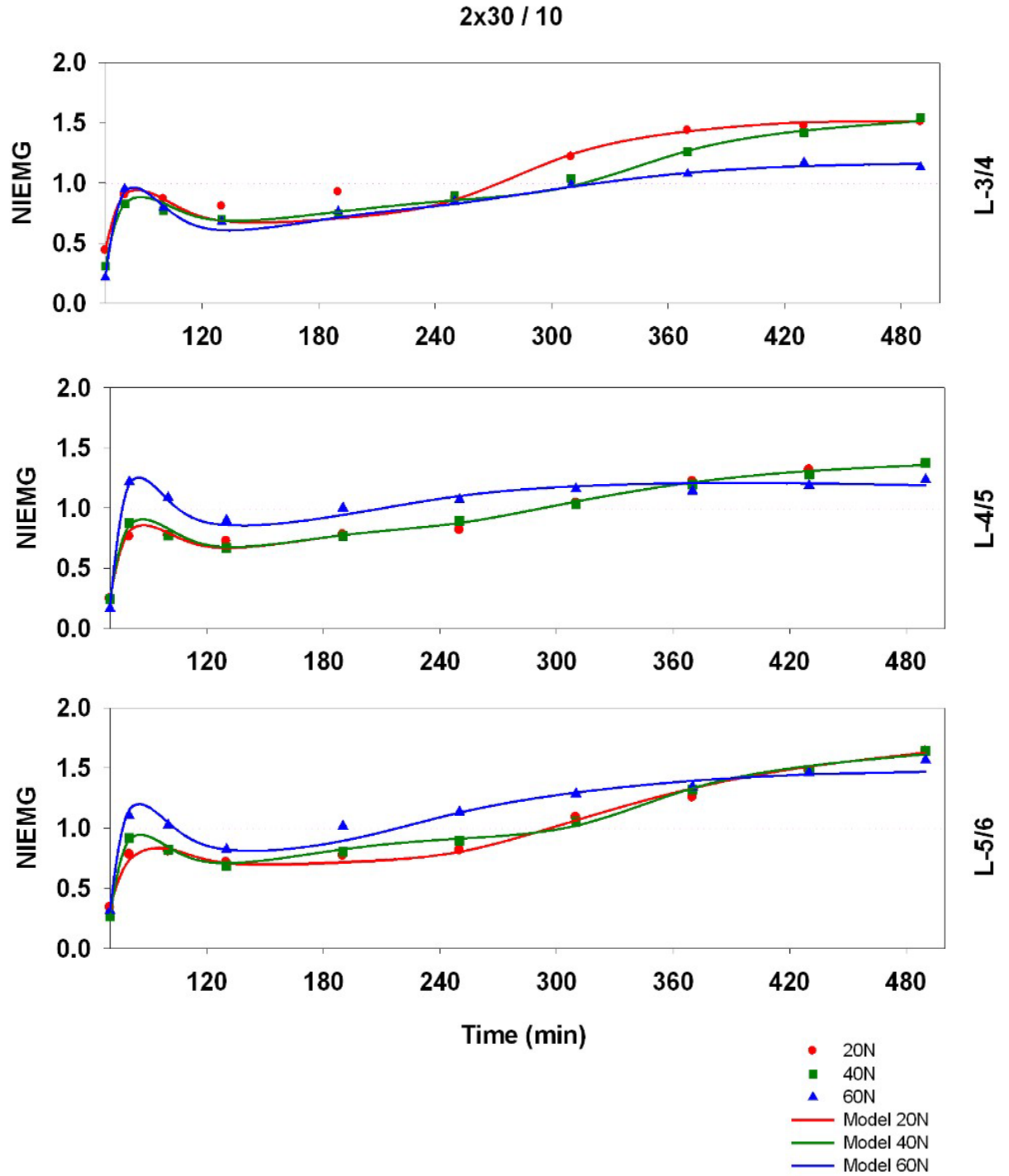


Figure 18. Mean NIEMG data and the developed models for the 7 h recovery period are superimposed for 20, 40 and 60N loads.

Inflammation is a defense reaction caused within as a response to tissue damage or injury, which may be the result of physical trauma. The primary objective of inflammation is to isolate the damaged area, mobilize effector cells and molecules to the damaged site, and ultimately promote healing. An inflammatory reaction will respond to tissue damage within hours of the occurrence. Once the initial immune reaction is activated, a cascade of other reactions follows. The inflammatory response will continue until the tissue is healed. Increased exposure time to a certain exercise leads to a greater inflammatory response (Leadbetter, 1990). NIEMG was observed to be greater than 1.0 in all lumbar levels in all preparations at the end of recovery. Although it is known that inflammation and therefore, delayed hyperexcitability, gradually decrease with rest, the effects of inflammation were still observed at the end of the 7 hour recovery period.

Two significant conclusions can be derived from the results of this study. A full neuromuscular disorder resulted from static exposure to a 3:1 work-to-rest duration ratio, regardless of load magnitude. Therefore, it is apparent that a longer continuous duration of load exposure is the primary factor in promoting a neuromuscular disorder, with load magnitude being of secondary importance. The experimental evidence also suggests that after a period of time the effects of load duration will override the effects of load magnitude.

These conclusions deviate somewhat from the conclusions of previous studies. Recently, it has been shown that load magnitude significantly affects the manifestation components of a neuromuscular disorder, namely initial hyperexcitability. A cumulative loading period of 60 min (an equivalent duration to the current study) comprised of 6 intervals of 10 min work periods followed by 10 min rest periods (a 1:1 work-to-rest ratio) did not elicit an initial hyperexcitability peak above the 1.0 preload value (Sbriccoli et al., 2004). Therefore, load

magnitude presumably has an impact on the initial hyperexcitability component of neuromuscular disorder magnitude for shorter periods of static load exposure. Previous research has shown that the degree of manifestation of delayed hyperexcitability closely corresponds to load magnitude or to the overall time of loading (Sbriccoli et al., 2004; Sbriccoli et al., In Press). This study supports the idea that overall time of loading has a greater degree of influence than load magnitude on delayed hyperexcitability development. The differentiation between results confirms that different work-to-rest ratio influence neuromuscular disorder development and influences the onset of a cumulative low back disorder.

The body's response to the condition of lumbar static flexion constitutes a neuromuscular disorder although the disorder is transient. Spasms and muscular hyperexcitability are established responses to tissue damage and are indicative of low back disorders (Fisher and Chang, 1985; Hoyt et al., 1981; Miller, 1985). Although the components of this disorder are transient, long-term exposure to risk factors such as static or cyclic flexion will expose the spine to the cumulative effects of viscoelastic creep. Workers that are required to repeatedly place stress on their lower backs via repetitive bouts of sustained flexion with little rest are at risk to develop residual creep, microdamage, and acute inflammation. An overnight rest of 7 hours is not adequate for full recovery of creep or inflammation resulting in the presence of residual creep at the start of the next workday. As this cycle is repeated and the tissues and joints of the lumbar spine are continually subjected to trauma and cumulative wear and tear, acute inflammation will progress to chronic inflammation and viscoelastic tissues may lose the ability to recover to their original resting length. Microtrauma healing must exceed microtrauma production in order to avoid chronic inflammation and the associated pain, muscle stiffness, weakness, and limited range of motion (Leadbetter, 1990; Safran, 1985).

It is important to note that the results obtained from the feline model used in the present study are not directly transferable to humans. However, research has established that humans respond similarly to static and cyclic ligament loading. The presence of the ligamento-muscular reflex in the human lumbar spine elicited spasms and altered muscular activity postflexion in human subjects performing 10 minutes of continuous static flexion (Solomonow et al., 1985; Solomonow et al, 2003a). Therefore, valuable insight into human responses to certain conditions can be gained from the feline model.

In conclusion, a cumulative low back disorder was elicited from exposure to two 30 min static loads spaced by a 10 min interval. Although the valuable effects of rest have been documented (Sbriccoli, 2004), a 3:1 work-to-rest duration ratio was not sufficient to attenuate or prevent the development of any of the components of a neuromuscular disorder and the associated microdamage and inflammation.

An extension of this research would be to further investigate various work-rest cycles. First, the physiological phenomenon that occurs in work-rest cycles of the same cumulative time (70 min) but different work and rest exposure times must be observed. Then, the results from the various work-rest durations must be compared to determine which will result in the least amount of damage and result in the quickest recovery. It is imperative to observe this phenomenon in the feline model, but to also supplement this data with experimental data from humans.

Another research possibility would be to perform a similar investigation in which the physiological behavior for different work-rest cycles with varying load magnitudes and load durations is observed in preparations undergoing cyclic loading. In the future, the combined effects of load magnitude, load duration, number of repetitions, and the work-to-rest duration

ratio as a whole should be considered in order for an optimal dose-duration ratio to be designed for static and cyclic loading.

REFERENCES

- Acierno, S., Baratta, R., Solomonow, M. (1995). A Practical Guide to Electromyography for Ergonomists and Biomechanists. Louisiana State University.
- Adams, M.A. and Dolan, P. (1995). Recent advances in lumbar spinal mechanics and their clinical significance. *Clinical Biomechanics*, **10**(1), 3-19.
- Adams, M.A., Hutton, W.C., and Scott, J.E.E. (1980). The resistance to flexion of the lumbar intervertebral joint. *Spine*, **5**(3), 245-253.
- Basmajian, J.V. and DeLuca, C.J. (1986). Muscles Alive: Their functions revealed by electromyography. 5th edition. Baltimore: Williams and Wilkins.
- Burdorf, A., Govaert, G., and Elders, L. (1991). Postural load and back pain of workers in the manufacturing of prefabricated concrete elements. *Ergonomics*, **34**(7), 909-918.
- Bureau of Labor Statistics. (2003). Workplace Injuries and Illnesses, Report USDL-03-138. U.S. Department of Labor, Washington, DC.
- Carpenter, J.E., Flanagan, C.L., Thomopoulos, S., Yian, E.H., and Soslowsky, L.J. (1998). The effects of overuse combined with intrinsic or extrinsic alterations in an animal model of rotator cuff tendinosis. *Am J Sports Med*, **26**(6): 801-807.
- Cholewicki, J. and McGill, S. (1991). Lumbar spine load during lifting of extremely heavy weights. *Med Sci Sports Exerc*. **23**(10), 1179-1186.
- Claude, L.N., Solomonow, M., Zhou, B.H., Baratta, R.V., and Zhu, M.P. (2003). Neuromuscular dysfunction elicited by cyclic lumbar flexion. *Muscle Nerve*, **27**(3), 348-358.
- Crisco, J., Panjabi, M., Yamamoto, I., Oxland, T. (1992). Stability of the human ligamentous spine: II. Experiment. *Clinical Biomechanics*, **7**, 27-32.
- Damkot, D.K., Pope, M.H., Lord, J., and Frymoyer, J.W. (1984). The relationship between work history, work environment and low-back pain in men. *Spine*, **9**(4), 395-399.
- Dorland's Medical Dictionary. (1994). 28th Edition, Philadelphia: W.B. Saunders Company, ISBN: 0-7216-2859-1.
- Elders, L.A. and Burdorf, A. (2001) Interrelations of risk factors and low back pain in scaffolders. *Occup Environ Med*, **58**(9): 597-603.
- Eversull, E., Solomonow, M., Zhou, B.H., Baratta, R.V., and Zhu, M.P. (2001). Neuromuscular zones sensitivity to lumbar displacement rate. *Clinical Biomechanics*, **16**, 102-113.

Hoyt, W., Hunt, H., and DePauw, M. (1981). EMG assessment of chronic low back pain syndrome. *J Am Osteopath Assoc*, **80**: 728-730.

Field, H. and Taylor, M. (1992). An atlas of cat anatomy. Second edition. The University of Chicago Press.

Fisher, A. and Chang, C. (1985). Electromyographic evidence of paraspinal muscle spasms during sleep in patients with low back pain. *Clin J Pain*, **1**, 147-154.

Frazer, M., Callaghan, J., Marras, W., and McGill, S. Cumulative load: toward the metric for optimal occupational health – a low back example. *Human Factors*, **In Press**.

Ganong, W.F. (1981). Review of Medical Physiology. Tenth Edition. Los Altos: Lange.

Garg, A. and Moore, J.S. (1992). Epidemiology of low-back pain in industry. *Occup Med*, **7**(4): 593-608.

Gedalia, U., Solomonow, M., Zhou, B., Baratta, R., and Harris, M. (1999). Biomechanics of increased exposure to lumbar injury caused by cyclic loading: Part 2. Recovery of reflexive muscular stability with rest. *Spine*, **24**(23), 2461-2467.

Granata, K. and Marras, W. (1995). The influence of trunk muscle coactivity on dynamic spinal loading. *Spine*, **20**(8): 913-919.

Gray, Henry F.R.S. (1974). Gray's Anatomy, Descriptive and Surgical. Philadelphia: Running Press.

Guanche, C., Knatt, T., Solomonow, M., Lu, Y., and Baratta, R. (1995). The synergistic action of capsule and the shoulder muscles. *Am J Sports Med*, **23**(3), 301-306.

Guo, H.R. (2002). Working hours spent on repeated activities and prevalence of back pain. *Occup Environ Med*, **59**(10), 680-688.

Hirokawa, S., Solomonow, M., Luo, Z., Lu, Y., D'Ambrosia, R. (1991). Muscular co contraction and control of knee stability. *Journal of Electromyography and Kinesiology*, **1**, 199-208.

Hirsch, C., Inglemark, B., Miller, M. (1963). The anatomical basis for low back pain: study on the presence of sensory nerve endings in ligaments, capsular, and disc structures in the human lumbar spine. *Acta Orthopaedica Scandinavica*, **33**:1-17.

Holm, S., Indahl, A., Solomonow, M. (2002). Sensorimotor control of the spine. *Journal of Electromyography and Kinesiology*, **12**(3), 219-234.

Hoogendoorn, W.E., Bongers, P.M., de Vet, H.C., Douwes, M., Koes, B.W., Miedema, M.C., Ariens, G.A., and Bouter, L.M. (2000). Flexion and rotation of the trunk and lifting at work are risk factors for low back pain: results of a prospective cohort study. *Spine*, **25**(23), 3087-3092.

Jackson, M., Solomonow, M., Zhou, B., Baratta, R. and Harris, M. (2001). Multifidus EMG and Tension-Relaxation Recovery After Prolonged Static Lumbar Flexion. *Spine*, **26**(7), 715-723.

Kaigle, A., Holm, S., and Hansson, T. (1995). Experimental instability in the lumbar spine. *Spine*, **20**(4), 421-430.

Kerr, M.S., Frank, J.W., Shannon, H.S., Norman, R.W., Wells, R.P., Neumann, W.P., and Bombardier, C. (2001). Biomechanical and psychosocial risk factors for low back pain at work. *Am J Public Health*, **91**(7), 1069-1075.

Knatt, T., Guanche, C., Solomonow, M., Lu, Y., Baratta, R., Zhou, B.H. (1995). The glenohumeral biceps reflex in the feline. *Clinical Orthopaedics*, 314, 247-252.

Kumar, S. (2001a). Cumulative load as a risk factor for back pain. *Spine*, **15**(12), 1311-1316.

Kumar, S. (2001b). Theories of musculoskeletal injury causation. *Ergonomics*, **44**(1), 17-47.

Leadbetter, W. (1990). An introduction to sports induced soft tissue inflammation. In: *Sports Induced Inflammation*, edited by Leadbetter, W., Buckwalter, J., and Gordon, S. Park Ridge, IL: AAOS.

Lewis, J., Donatto, K., Solomonow, M., Zhou, B.H., Lu, Y., Baratta, R., Zhu, M.P. (1996). Ligamento-muscular protective reflex in the feline ankle. *Orthopaedics International*, **4**, 359-365.

McGill, S.M. and Norman, R. (1986). Partitioning of the L4-L5 dynamic moment into disc, ligamentous, and muscular components during lifting. *Spine*, **11**(7): 666-668.

Miller, D. (1985). Comparison of EMG activity in the lumbar paraspinal muscles of subjects with and without chronic low back pain. *Phys Ther*, **65**: 1347-1354.

National Academy of Sciences. (1999). *Work Related Musculoskeletal Disorders: Report, Workshop Summary, and Workshop Papers*. Washington, DC. pp.1-240.

National Institute of Occupational Safety and Health. (1997). *Musculoskeletal Disorders (MSDs) and Workplace Factors*.

National Occupational Research Agenda. (1997). *Low Back Disorders Additional Information*. National Institute of Occupational Safety and Health.

National Research Council. (2001). *Musculoskeletal disorders and the workplace: Low back and upper extremities*. Panel on Musculoskeletal Disorders and the Workplace, Commission on Behavioral and Social Sciences and Education, National Research Council and Institute of Medicine. National Academy of Science, Washington, DC. pp.1-512.

Neuromuscular Research Center (2002). Motor Unit Action Potential. Online. Internet. May 10, 2004. <http://www.nmrc.bu.edu/tutorials/motor_units/decomp.html>

Norman, R., Wells, R., and Neumann, P. (1998). Comparison of peak vs. cumulative work exposure risk factors for the reporting of low back pain in the automotive industry. *Clin Biomech*, **13**(8), 561-573.

Occupational Safety and Health Administration. (2000). Ergonomics program, Final Rule: 29 CFR Part 1910. Department of Labor, *Inflammation*, **64**: 68262-68870.

Panjabi, M. and Courtney, T. (2001). High speed subfailure stretch of rabbit anterior cruciate ligament: change in elastic failure and viscoelastic characteristics. *Clinical Biomechanics*, **16**(4), 334-340.

Panjabi, M. (1996). Low Back Pain, A Scientific and Clinical Overview. In: Weinstein, J., Gordon, S., eds. Low Back Pain. Rosemont, IL: American Academy of Orthopedic Surgeons, 367-384.

Panjabi, M. (1993). Spinal Instability. In: Contemporary Perspectives in Neurosurgery. Springer Verlag.

Panjabi, M.M. (1992). The stabilizing system of the spine: I. Function, dysfunction, adaptation, and enhancement. *Journal of Spinal Disorder*, **5**(4):383-389.

Paton, W.D., and Wand, D.R. (1967). The Margin of Safety of Neuromuscular Transmission. *Journal of Physiology*, **191**, 59-90.

Petrie, S., Collins, G., Solomonow, M., Wink, C., Chuinard, R., D'Ambrosia, R. (1998). Mechanoreceptors in the human elbow ligaments. *The Journal of Hand Surgery*, **23A**(3), 512-518.

Phillips, D., Petrie, S., Solomonow, M., Zhou, B.H., Guanche, C., D'Ambrosia, R. (1997). Ligamento-muscular protective reflex in the elbow. *The Journal of Hand Surgery*, **22A**(3), 512-518.

Pope, M., Andersson, G., Broman, H., Svensson, M., and Zetterberg, C. (1986). Electromyographic studies of the lumbar trunk musculature during development of axial torque. *Journal of Orthopaedic Research*, **4**(3), 288-297.

Posner, I., White, A., Edwards, T., and Hayes, W. (1982). A biomechanical analysis of the clinical stability of the lumbar and lumbosacral spine. *Spine*, **7**(4), 374-389.

Punnett, L., Fine, L., Keyserling, W., Herrin, G., and Chaffin, D. (1991). Back disorders and nonneutral trunk postures of automobile assembly workers. *Scand J Work Environ Health*, **17**(5), 337-346.

Radwin, R., Marras, W., and Lavender, S. (2002). Biomechanical aspects of work-related musculoskeletal disorders. *Theoretical Issues in Ergonomics*, **2**(2), 153-217.

Rhalmi, W., Yahia, H., Newman, N., Isler, M. (1993). Immunohistochemical study of nerves in lumbar spine ligaments. *Spine*, **18**(2), 264-267.

Rosse, C. and Gaddum-Rosse, P. (1997). Hollinshead's Textbook of Anatomy. 5th Edition. Philadelphia: Lippincott Raven Publishers.

Safran, M. (1985). Elbow injuries in athletes. *Clinical Orthopedics*, **310**: 257-277.

Sbriccoli, P., Solomonow, M., Zhou, B.H., Baratta, R.V., Lu, Y., Zhu, M.P., and Burger, E.L. (2004). Static load magnitude is a risk factor in the development of cumulative low back disorder. *Muscle Nerve*, **29**(2), 300-308.

Sbriccoli, P., Yousuf, K., Kupershtein, I., Solomonow, M., Zhou, B.H., Zhu, M.P., and Lu, Y. Static repetition is a risk factor in the development of lumbar cumulative musculoskeletal disorder. *Spine*, **In Press**.

Sihvonen, T., Partanen, J., Hanninen, O., Soimakallio, S. (1991). Electric behavior of low back muscles during lumbar pelvic rhythm in low back pain patients and healthy controls. *Arch Phys Med Rehabil*, **72**(13), 1080-1087.

Silverstein, B., Fine, L., and Armstrong, T. (1986). Hand wrist cumulative trauma disorders in industry. *Br J Ind Med*, **43**(11): 779-784.

Solomonow, M., Baratta, R.V., Banks, A., Freudenberger, C., and Zhou, B.H. (2003a). Flexion-relaxation response to static lumbar flexion in males and females. *Clin Biomech*, **18**(4), 273-279.

Solomonow, M., Baratta, R.V., Zhou, B.H., Burger, E., Zieske, A., and Gedalia, A. (2003b). Muscular dysfunction elicited by creep of lumbar viscoelastic tissue. *Journal of Electromyography and Kinesiology*, **13**, 381-396.

Solomonow, M., Hatipkarasulu, S., Zhou, B.H., Baratta, R.V., and Aghazadeh, F. (2003c). Biomechanics and electromyography of a common idiopathic low back disorder. *Spine*, **28**(12), 1235-1248.

Solomonow, M., Zhou, B.H., Baratta, R.V., and Burger, E. (2003d) Biomechanics and electromyography of a cumulative lumbar disorder: response to static flexion. *Clinical Biomechanics*. **18**: 890-898.

Solomonow, M., Zhou, B., Baratta, R.V., Zhu, M., Lu, Y. (2002). Neuromuscular disorders associated with static lumbar flexion: a feline model. *Journal of Electromyography and Kinesiology*, **12**(2), 81-90.

- Solomonow, M., Zhou, B.H., Baratta, R., Lu, Y., Zhu, M., and Harris, M. (2000). Biexponential recovery model of lumbar viscoelastic laxity and reflexive muscular activity after prolonged cyclic loading. *Clinical Biomechanics*, **15**(3), 167-175.
- Solomonow, M., Zhou, B.H., Baratta, R., Lu, Y., Harris, M. (1999). Biomechanics of increased exposure to lumbar injury caused by cyclic loading: Part I. Loss of reflexive muscular stabilization. *Spine*, **24**(23), 2426-2434.
- Solomonow, M., Zhou, B., Harris, M., Lu, Y., and Baratta, R. (1998). The ligamento-muscular stabilizing system of the spine. *Spine*, **23**(23), 2552-2562.
- Solomonow, M., Guanche, C., Wink, C., Knatt, T., Baratta, R., Lu, Y. (1996). Mechanoreceptors and reflex arc in the feline shoulder. *J Elbow Shoulder Surgery*, **5**, 139-146.
- Solomonow, M., Baratta, R.V., Zhou, B., Shoji, H., Bose, W., Beck, C., D'Ambrosia, R. (1987). The synergistic action of the ACL and thigh muscles in maintaining joint stability. *Am J Sports Med*, **15**(3), 207-213.
- Sommerich, C.M., McGlothlin, J.D., and Marras, W.S. (1993). Occupational risk factors associated with soft tissue disorders of the shoulder: a review of recent investigations in the literature. *Ergonomics*, **36**(6), 697-717.
- Soslowsky, L.J., Thomopoulos, S., Tun, S., Flanagan, C.L., Keefer, C.C., Mastaw, J., and Carpenter, J.E. (2000). Neer Award 1999. Overuse activity injures the supraspinatus tendon in an animal model: a histologic and biomechanical study. *J Shoulder Elbow Surg* **9**(2), 79-84.
- Stubbs, M., Harris, M., Solomonow, M., Zhou, B., Lu, Y., Baratta, R. (1998). Ligamento-muscular protective reflex in the lumbar spine of the feline. *Journal of Electromyography and Kinesiology*, **8**(4), 197-240.
- Teo, E.C. and Ng, H.W. (2001). Evaluation of the role of ligaments, facets and disc nucleus in lower cervical spine under compression and sagittal moments using finite element method. *Med Eng Phys*, **23**(3), 155-164.
- Thorbjornsson, C.B., Alfredsson, L., Fredriksson, K., Michelsen, H., Punnett, L., Vingard, E., Torgen, M., and Kilbom, A. (2000). Physical and psychosocial factors related to low back pain during a 24-year period. A nested case-control analysis. *Spine*, **25**(3), 369-374; discussion 375.
- Thornton, G.M., Shrive, N.G., and Frank, C.B. (2003). Healing ligaments have decreased cyclic modulus compared to normal ligaments and immobilization further compromises healing ligament response to cyclic loading. *J Orthop Res*, **21**(4), 716-722.
- Twomey, L.T., Taylor, J.R., Oliver, M.J. (1988). Sustained flexion loading, rapid extension loading of the lumbar spine and the physical therapy of related injuries. *Physiother Pract*, **4**, 129-138.

- van Dieen, J.H., Cholewicki, J., Radebold, A. (2003). Trunk muscle recruitment patterns in patients with low back pain enhance the stability of the lumbar spine. *Spine*, **28**(8), 834-41.
- White, A.A. III, and Panjabi, M.M. (1978). *Clinical Biomechanics of the Spine*. Philadelphia: JB Lippincott.
- Williams, M., Solomonow, M., Zhou, B.H., Baratta, R.V., and Harris, M. (2000). Multifidus spasms elicited by prolonged lumbar flexion. *Spine*, **25**(22): 2916-2924.
- Wirth, O. and Cutlip, R.G. (2001). Animal models for controlling and quantifying voluntary muscle performance of rats using operant conditioning. *Advantages in Occupational Ergonomics and Safety*. IOS Press. ISBN: 158603 174 0.
- Woo, S., Aprelena, M., Hoher, J. (1999). Tissue mechanics of ligaments and tendons. In: Kumar, S., editor. *Biomechanics and ergonomics*. Philadelphia: Taylor & Francis.
- Wooburne, R.T. and Burkel, W.E. (1988). *Essential of Human Anatomy*. 8th Edition. New York: Oxford University Press, Inc.
- Yahia, H. and Newman, N., 1993. A scanning electron microscopic and immunohistochemical study of spinal ligaments innervation. *Ann Anat*, **175**(2), 111-114.
- Yahia, H. and Newman, N. (1991). Innervation of spinal ligaments of patients with disc herniation. *Pathological Research Practice*, **187**(8), 936-938.
- Yahia, H., Newman, N., and Richard, C. (1988). Neurohistology of lumbar spine ligaments. *Acta Orthopaedica Scandinavica*, **59**(5), 508-512.

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